ICAL
TROCARDIOGRAPHY.

THOMAS LEWIS

CLINICAL ELECTROCARDIOGRAPHY

BY

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LONDON:

SHAW & SONS, 7 & 8, FETTER LANE, E.C.

Printers and Publishers.

1913.

LONDON:

PRINTED BY SHAW AND SONS, FETTER LANE, E.C.

PREFACE.

IN offering to members of the medical profession an account of clinical electrocardiography, I do so with the conviction that this new method of examination has become essential to the modern diagnosis and treatment of cardiac patients. When some four years ago I commenced to study disorders of the heart with the aid of the "string galvanometer" the method was in its early infancy; although it was regarded at that time as full of promise, yet its scope, in helping to perfect our acquaintanceship with heart disease, could not then be foreseen. Neither can we see at the present time how far it may still lead us; it has already taken us far. Electrocardiography has begun to fill the great gap in our knowledge of these maladies; for it is a direct means of examining the all essential heart muscle.

In writing a treatise upon the "*Mechanism of the Heart Beat*," my subject entailed a free use of electro-cardiographic illustration, and thus compelled a brief description of the method; for at the time no adequate account of it was extant in the English language. To instruct its readers in the use of the galvanometer or to acquaint them with the clinical application of its records, was not the primary object of that book. It dealt with these subjects so far as was essential to prove and explain the mechanism of disordered heart action. My later purpose has been to describe the manner in which abnormal forms of heart action are recognised, and to relate their significance. In "*Clinical Disorders of the Heart Beat*" I have attempted to collect the more simple bedside signs and to narrate their influence upon prognosis and treatment in a manner palatable to the pure clinician. The present chapters are intended as a supplement to that book. They describe the most precise method which we possess when we attempt to identify the several disorders of heart action already spoken of in "*Clinical*

Disorders." It frequently happens that other graphic records fail us in analysing these the common disturbances; electrocardiography is the last court of appeal and its judgment is rarely at fault. There are certain abnormal types of heart action upon which other methods are almost, if not quite, silent; knowledge of these conditions is in the almost exclusive possession of electrocardiography; when such are described I shall refer briefly to the manner in which they affect the management of the patient. Finally, the electric curves have revealed a number of new physical signs, associated with the abnormal distribution and arrangement of the muscular masses in the heart. These will be discussed.

Those cardiac patients are few, in whom an electric examination is superfluous, and in a large and increasing percentage of cases the records profoundly modify our conception of the conditions with which we deal. The time is not distant, when no hospital which undertakes the care of many of these patients may neglect the string galvanometer, if

it is to rank amongst institutions whose design is proficiency.

Electrocardiography has developed; it has grown along technical lines; it has embraced a terminology of its own, the inevitable result of progress in a new direction. It is doing its share in increasing the already overweighted burden of general medicine. We may not deplore, but must welcome the fullness of this load; recognising in it an increase of our capacity.

T. L.

27, *Queen Anne Street,*
Cavendish Square,
November, 1912.

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CHAPTER I.

THE ELECTROCARDIOGRAPHIC METHOD.

That electric currents are developed in the heart at each contraction of this organ was demonstrated by Kölliker and Müller as early as the year 1856. Modern electrocardiography is the outcome of this discovery. The currents are small, yet modern instruments are sufficiently sensitive to record them with facility. It is even unnecessary that the heart should be exposed ; the currents will move the recording fibre of a suitable galvanometer when the latter is connected to the limbs of the human subject. It is the study of the direction, time-relations and magnitude of these currents which constitutes modern electrocardiography.

The instrument which is described in the present chapter is known as the "string galvanometer," the invention of Einthoven, a Dutch physiologist. It consists essentially of a powerful electro-magnet, the poles of which are closely approximated (Fig. 1) ; and of an extremely delicate conducting fibre of silvered quartz or glass which is stretched in the narrow gap between the two magnetic poles. If minute currents are led through this fibre, as it lies in the magnetic field, the fibre moves in response to the tested current in a plane at right angles to the lines of magnetic force across the gap. The magnified shadow of the

fibre is vertical and is projected by means of an optical system and powerful light on to the horizontal slit of a camera ; the shadow moves at right angles to and across the slit and the movements are photographed upon a sensitive plate which travels behind the slit. I do not propose to describe the detailed construction of the galvanometer itself or of

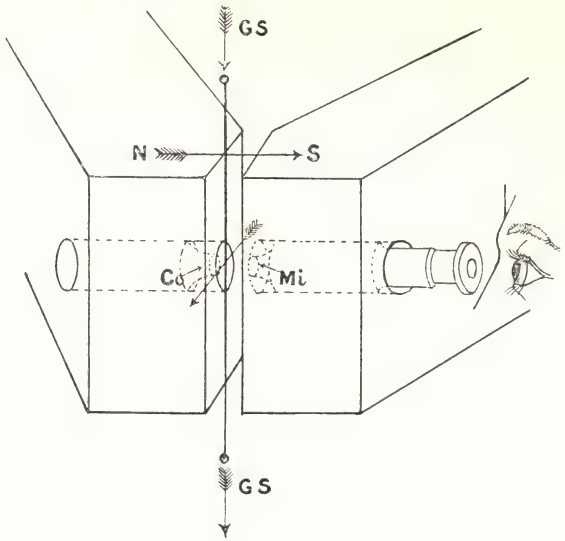

Fig. 1. A diagram illustrating the construction of the string galvanometer. The poles of the magnet (*N*, *S*) are seen in outline ; the recording fibre (*GS*) lies vertically between them ; its movements may be observed through a microscope (*Mi*). The movements of the string are in a plane parallel to the faces of the magnetic poles, as indicated by the central arrow. *Co* is a condenser.

the accessory apparatus which it is necessary to employ in obtaining human electrocardiograms. Many different installations are now sold and some are specially arranged for clinical purposes. The galvanometer illustrated in Fig. 2 is perhaps the most serviceable of any, and is to be recommended for the simplicity of its construction and the ease with which it is kept in order and manipulated. The

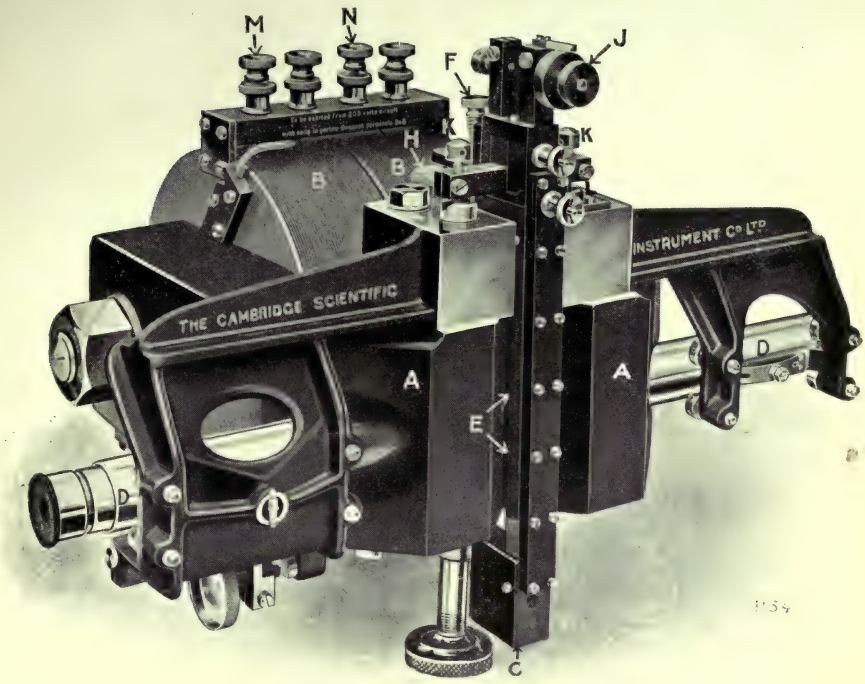

Fig. 2. The string galvanometer, as modelled by the Cambridge Scientific Instrument Company. The coils of the magnet (*B*, *B*) are seen in the background; they are supplied with current through the four terminals (*M*, *N*); the poles of the magnet (*A*) are in the foreground. The magnet is pierced by microscope and condenser (*D*, *D*). The string is encased in a carrier (*E*), which is suspended from above upon two knife edge rests (*K*, *K*) and hangs between the magnetic poles. The string tension is adjusted by means of the milled screw (*J*); the position of the carrier in the field is altered by an adjustment at *F*. The tested current is led to the instrument through the two terminals which are seen on the carrier directly below the screw *J*.

adjustments are simple and consist of mill-headed screws, which level the instrument, centre the fibre in the field, increase or decrease its tension, and focus its shadow upon the camera. Those who desire a more detailed account of the apparatus may refer to the special descriptions issued

by its makers; but familiarity with galvanometric outfits can be gained only by intimate acquaintanceship with working instruments.

The galvanometer is connected to the patient through some special form of switchboard. Students of electrocardiography utilise switchboards of different patterns, but I shall confine myself to a description of a relatively simple

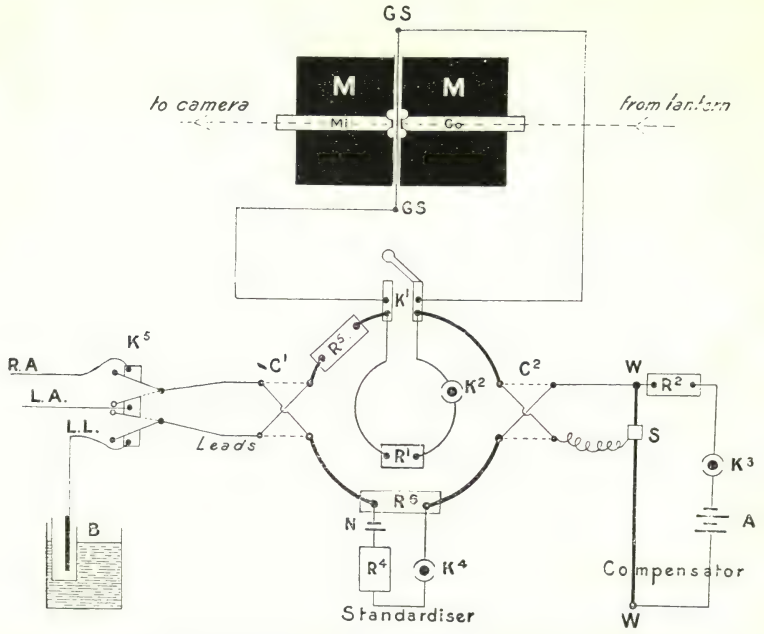

Fig. 3. A scheme of the connections used for routine clinical work, as it is installed at University College Hospital Medical School. The scheme is fully described in the text.

scheme which is serviceable for routine clinical observation. Fig. 3 is a diagram of the apparatus and its connections. The string of the galvanometer (*GS*) is connected to a key (K^1) which closes the string circuit. The closure of this key brings the string to rest and safeguards it from damage. The same key communicates with two circuits, the inner

and outer circles of the diagram; the former is a shunt, containing a resistance and key (R^1 and K^2), so arranged that when closed it carries the greater part of the tested current, thus preserving the string from mishap. It is used when a current of unknown strength is thrown into the string circuit. The outer circle represents the main circuit; it is broken at three points; (1) at K^1 where it joins the string circuit, (2) at C^1, by a commutator, where it connects to the patient and (3) at C^2, by a commutator, where it connects to the compensator. It contains also, a dial resistance (R^5) and the standardiser. The separate parts of the apparatus may be described briefly and in rotation.

Connection of patient. One wire which runs from the commutator C^1 to the key K^5 may be connected at will to the right arm (*R. A.*) or left arm (*L. A.*) by means of the special key; the second or parallel wire may be connected to the left arm or left leg (*L. L.*) by the same key.* The actual contacts are made when the limbs are immersed in salt water, and through porous pots containing zinc sulphate (Fig. 3 *B* and Fig. 4). The key (K^5) is arranged so that the two main wires may have the following paired connections.

	Limbs connected.
Lead *I*	*R. A.* and *L. A.*
Lead *II*	*R. A.* and *L. L.*
Lead *III*	*L. A.* and *L. L.*
Short circuit	*L. A.* and *L. A.*

These are the connections which are employed in routine observation. The commutator serves to reverse the direction of the leads, and is rarely employed.

* The wires which connect to the limbs may be of any length; the patient who is observed may be in a separate building.

Fig. 4. Photograph of a subject as connected for observation. The two arms and the left leg are used, and curves are taken from the three leads which are represented by the arrows drawn upon the figure. The zinc sulphate is placed in the outer vessels of the electrodes shown in this figure.

The compensatory circuit. This is shown to the right in Fig. 3 ; and consists of an accumulator (A), key (K^3), suitable resistance ($R^2 = 19$ ohms) and slide wire ($W\ W = 1$ ohm). It is used to bring the string shadow to zero when the patient is connected to the galvanometer ; it serves to neutralise the current derived from the skin, generally referred to as the

"skin current," and is fitted with a commutator, C^2, which reverses the direction of the compensatory current.

The standardiser is formed by a similar circuit to that of the compensator, but consists of a "Normal Weston cell," suitable resistances (R^4 and R^6) and key (K^4). It throws an E. M. F. of 3 millivolts* into the main circuit, and is used to standardise the string excursion, so that all electrocardiograms may be comparable one with another.

The dial resistance R^5 serves many purposes; amongst others, the measurement of the resistance of the string or of the patient's body.

The method of obtaining standardised electrocardiograms.

Standardised electrocardiograms are obtained in the following manner.

1. With the compensatory and standardising circuits open, but with the shunt (K^2) closed and the patient in circuit (K^5 connecting to *R. A.* and *L. A.*, the first lead), K^1 is opened. The string shadow immediately takes up a new position, as a result of skin current flowing into the galvanometer, and shows minute electrocardiograms (Fig. 5*a*).

2. K^3 is closed and a compensatory current, or current to balance the skin current, is introduced which brings the string shadow to zero once again (Fig. 5*b*).

3. The shunt (K^2) is opened and the string moves again† showing larger electrocardiograms (Fig. 5*c*), because the whole current is now allowed to flow through the string.

4. If the string shadow has moved much to one side, it is again brought to zero by moving the slider of the compensator.

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* Any number of millivolts, from 1 to 10, may be obtained.
† In Fig. 5 this movement is very small.

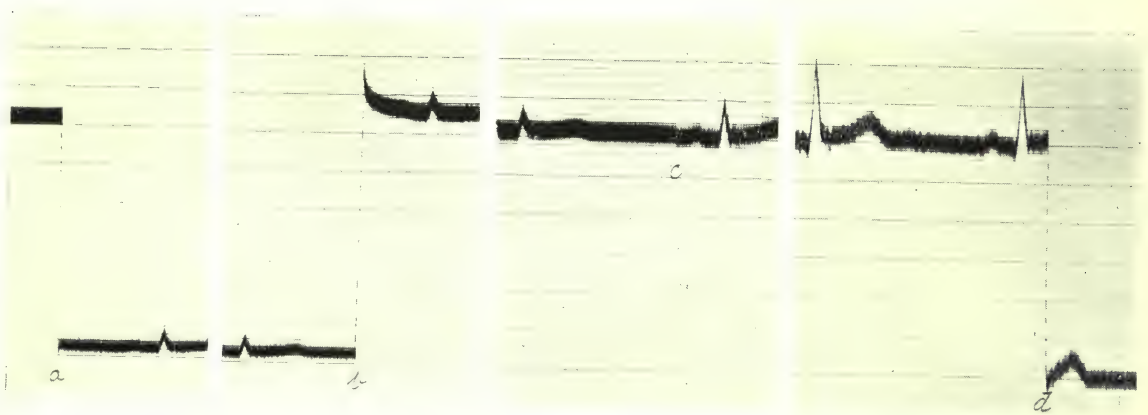


Fig. 5. Portions of a continuous curve, illustrating the steps taken in obtaining a standardised electrocardiogram. This and all other curves used as illustrations read from left to right. The string is at first still. At *a* the main key (K^1 of Fig. 3) is opened; the string moves in response to "skin current" and shows minute electrocardiograms. This current is compensated by throwing a current of opposite direction into circuit (*b*). At *c*, the shunt K^2 is removed, the string moves a little and the electrocardiogram increases in amplitude. The string tension is then adjusted until a potential of 3 millivolts deflects the whole curve through 3 centimetres (*d*). The curve is then a standard one and the photograph may be taken.

5. The standardising circuit (3 millivolts) is now made and broken repeatedly until, by adjusting the string tension, the whole curve is deflected through 3 centimetres (Fig. 5*d*). Tightening the string reduces and slackening increases the sensitiveness of the instrument.

6. A curve is taken from the first lead.

7. The process is repeated in the case of leads *II* and *III*. The curves obtained have the correct amplitude and a value of 3 millivolts to each 3 centimetres of excursion, or 1 millivolt to the centimetre of excursion.

For convenience of measurement, a screen, of millimetre lines, is placed immediately in front of the photographic plate. The light, falling through the screen, is intercepted by these lines and the film is ruled as the photograph is taken (Fig. 5).

Though the steps taken in producing standardised electrocardiograms may seem complicated and tedious in the description, yet in practice they become simple ; a series of curves from the three leads should be obtained from a single subject within a few moments.

Checking the accuracy of standardised curves.

The accuracy of excursion in a set of curves from a given subject may be checked in a simple manner by increasing the resistance in the main circuit. When curves have been taken from the three customary leads in the usual manner, a large resistance is placed in series with the string and patient by altering the dial resistance (R^5). As a rule it is convenient to throw in a resistance which is approximately equivalent to that of the string itself.* With this resistance

* The string resistance may be measured by obtaining a deflection of 6 centimetres with 3 millivolts, and by placing such a resistance in series with the string as halves its excursion ; *i.e.*, reduces it to 3 centimetres. The resistance of the string and the added quantity are then equal.

in circuit the same procedure is followed, the tension of the string being suitably decreased, so as to give the original excursion of 3 centimetres to 3 millivolts. The second group of curves, the one taken with the added resistance in series, should be similar in every respect to the original one.

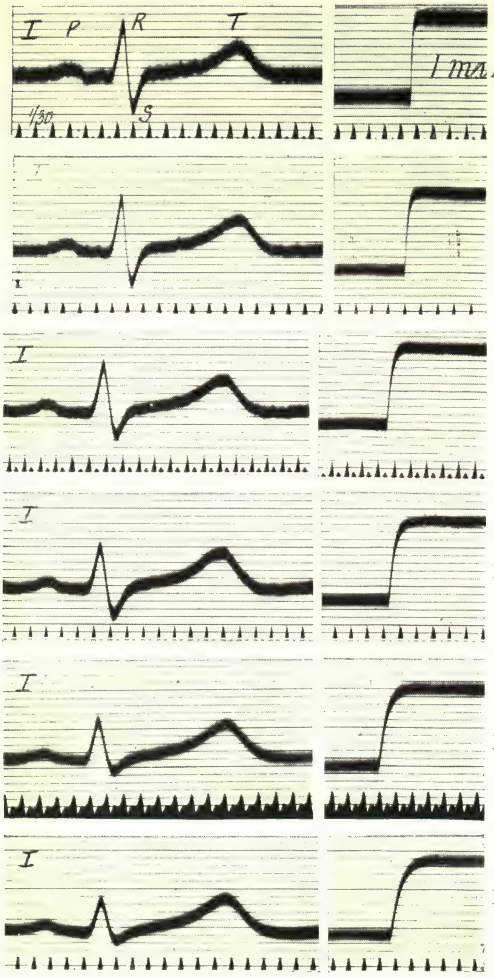


Fig. 6. Six electrocardiograms from lead *I* and from a single subject, and the six corresponding deflection curves. Illustrating the distortion of curves when the string tension is too slack. As the string is slackened beyond a certain limit and the deflection time (the time of response to an *E. M. F.* of 1 millivolt over an excursion of 1 centimetre) increases, *R* and *S* are materially reduced in amplitude. Time-marker in thirtieths of a second.

Testing certain properties of the string.

If standard curves, and curves of correct outline, are required, the response of the fibre to simple current changes should be carefully examined from time to time; for the excursion and shape of an electrocardiogram may be modified by the properties of the string. Supposing that an E. M. F. of 1 millivolt is thrown into circuit (the patient being disconnected) and that the tension of the fibre is arranged so

that it gives an excursion of 1 centimetre; the curve obtained has an outline similar to those shown to the right hand of Fig. 6. The string moves when the current enters it and eventually takes up a position 1 centimetre away. But in arriving at the new position it describes a curve. The characters of this curve are important. Fig. 6 shows six electrocardiograms from the same subject, and six responses to an E. M. F. of 1 millivolt. The curves differ because they were taken with different resistances in circuit, and consequently with different string tensions.* From above downwards the added resistances were increased and the string was therefore slackened. All the curves were taken with the string at such a tension that 1 millivolt gave 1 centimetre of excursion (see deflections to right of strips).

In the first place, the movement of the string in response to 1 millivolt should be "dead beat"; that is to say, there should be no over-shooting; over-shooting tends to produce distortion of the electrocardiograms by increasing the amplitude of the initial deflections. Over-shooting is shown in Fig. 5 (at *b*) at a stage when the tension of the fibre was too great. In the second place, the movement should be of sufficient rapidity. The slacker the string, the more slowly does it respond; the deflection times for the six strips of Fig. 6 are ·013, ·023, ·028, ·045, ·060 and ·070 seconds, respectively, from *A* to *F*. Now the initial electric changes which result from the heart beat are rapid, and if the quickest movement of which the string is capable is too slow to follow these changes, an accurate electrocardiogram is not obtained. Undue slackness of the string produces distortion of the curve and this distortion is well illustrated by the series of curves shown in Fig. 6. As the string is slackened, a time arrives when the steepest deflections (*R*, *S*) are shortened

* Adding resistance to the main circuit decreases the sensitiveness of the instrument and the string must be slackened to compensate this decrease.

and their upstrokes and downstrokes are rendered more oblique ; once distortion appears, further slackening exaggerates it ; so that, as in the last strips of Fig. 6, certain deflections (such as *S* and *R*) tend to disappear. The electrocardiograms of the first two strips are exactly alike, these electrocardiograms are accurate ; the first change, a decrease in the amplitude of *R* and *S* in the third strip, is seen when the deflection time of the string is increased to ·028 seconds.

For routine work the instrument and string should be tuned, so that while on the one hand there is no over-shooting, on the other hand the deflection time does not exceed ·02 seconds. The best strings for electrocardiographic work are those which give a considerable range, over which these two conditions are fulfilled.* Also it is necessary that the resistance of the string should be large compared with the resistance of the body, so that small variations of the latter do not appreciably affect the result. Strings having resistances of from 5,000 to 10,000 ohms are suitable.

* Intending students of electrocardiography are strongly advised to satisfy themselves fully of the adequacy in these respects of any instrument with which they propose to work.

CHAPTER II.

THE PHYSIOLOGICAL ELECTROCARDIOGRAM.

The electrocardiogram of a physiological heart beat consists of a series of deflections, some of which are rapid and of short duration while others are slow and of longer duration. They have been named in a purely empirical fashion, *P*, *Q*, *R*, *S* and *T*. The electrocardiogram opens with a blunt summit *P*, which occupies presystole, and is due to contraction of the two auricles (Fig. 7 and 8). Following upon this deflection the string shadow either maintains the zero position or dips somewhat. I speak of these portions of the electrocardiogram as the "auricular complex"; this complex begins with the upstroke of *P* and terminates at the opening of the "ventricular complex." The latter varies in the number of its component deflections; in its full form it comprises *Q*, a small downward deflection, *R* a steep, tall and pointed summit, *S* a steep depression of variable depth, and *T* a broad rounded summit. The period occupied by these deflections is approximately that of the ventricular systole to which they are due. The earliest sign of contraction in the ventricle occurs a little after the commencement of *R*, and usually during its upstroke. The contraction ends where *T* passes into the horizontal line of diastole, or within a few hundredths of a second of this point. These relations are indicated in Fig. 8, which is a simultaneous record of the electric changes and the apical heart sounds.

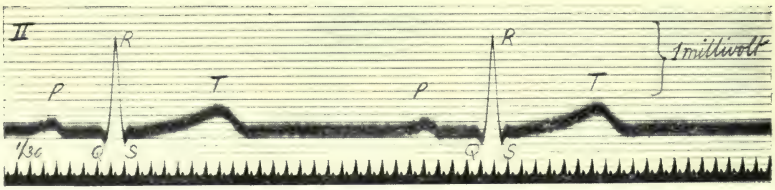


Fig. 7. An electrocardiogram from a normal subject, showing the auricular summit (*P*) and the ventricular deflections (*Q*, *R*, *S* and *T*).

In this and the remaining figures, the lead is marked in the upper left hand corner; the time, at the bottom, is in one-thirtieth sec., the ordinates each represent one-tenth millivolt.

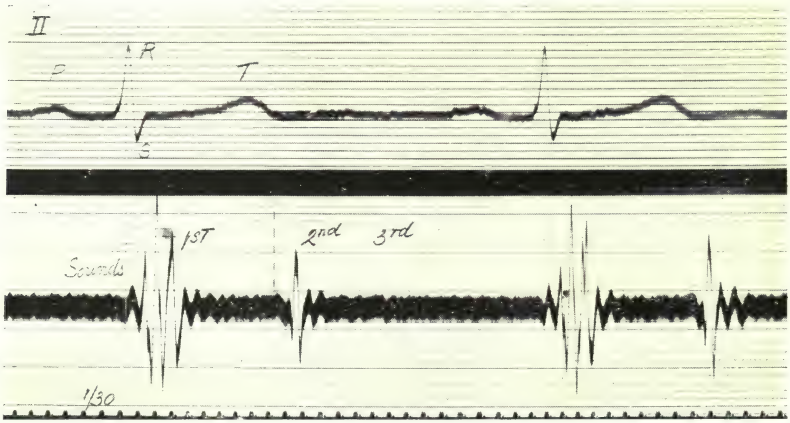


Fig. 8. Simultaneous electrocardiograms and heart sound curve from a normal subject. The figure shows the time-relations of the electrocardiogram to the beginnings of the 1st and 2nd heart sounds. All points on a vertical line are simultaneous.

There would be little gain at present in discussing the causation of the individual deflections of the electrocardiogram. We know that certain of them are auricular and that certain of them are ventricular; the curves provide us with clear indications of the systoles of auricles and of ventricles, and enable us to establish within very small limits of error,

the time-relations of contraction in upper and lower chambers of any given heart. In the mammalian electrocardiograms there is no evidence of a sinus contraction such as is present in the amphibia and reptilia (see Fig. 11*d*).

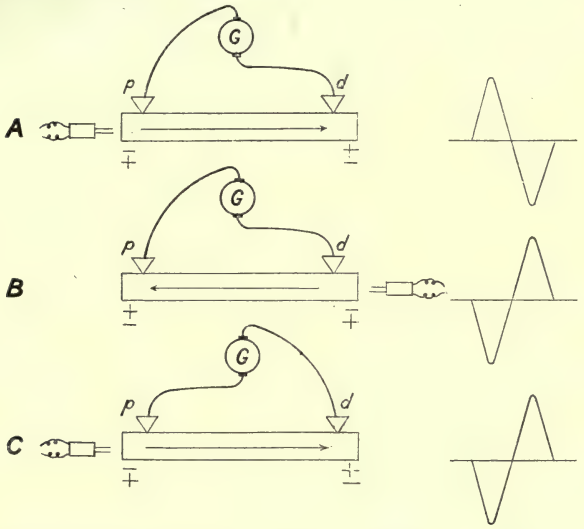


Fig. 9. A diagram illustrating the simple theory of electric curves. It represents a strip of somatic muscle connected to the galvanometer and stimulated to contract at one or other end. The deflections, which are shown to the right, vary in direction according to the point at which the contraction originates and with the relation of muscle to electrodes.

Supposing that we take a simple strip of muscle, the fibres of which are parallel, and connect its two ends, which we will term proximal (*p*) and distal (*d*), to the galvanometer. If this strip is stimulated by a single induction shock, say at its proximal end (Fig. 9*A*), then a wave of contraction flows from *p* to *d*. When the contraction begins at *p*, the muscle at this end becomes active, it also becomes relatively negative to *d*, as shown by the swing of the galvanometric recorder; the swing is in a definite and known direction, it is the same as that given when *p* is replaced by the zinc terminal of a

copper-zinc couple. But the wave of contraction passes to *d* and as it reaches this point it subsides at *p*. The distal end becomes active while the proximal end is resuming the quiescent state ; as an accompaniment of this change, *d* becomes relatively negative to *p*, and the swing of the recorder is reversed. Thus the whole electric effect consists of two deflections which are in opposite directions, a simple *diphasic* effect. Now if the stimulus is applied at the distal end (Fig. 9*B*) the direction of contraction is reversed ; under these circumstances *d* first shows relative negativity and later *p* ; a diphasic curve is still obtained, but the directions of swing are the reverse of those seen when the contraction travels from *p* to *d*. It will be evident that the recorded curve will also show reversed phases if, stimulation being at the proximal end, the connections to the galvanometer are reversed (Fig. 9*C*).

Thus experimental study teaches us that the shape of an electric curve is controlled by (1) the direction of the contraction wave in the muscle investigated, and this is governed by the point at which the contraction arises ; and (2) by the lie of this muscle in relation to the leading off electrodes. The full explanation of individual deflections in electrocardiograms is a difficult matter ; for the arrangement of the muscle fibres is very complicated and the contraction wave courses along them in a direction which changes from time to time and from point to point ; nevertheless, by arguing along general lines, we may formulate certain fundamental conclusions. It is known that all normal hearts yield, from a given lead, electrocardiograms which have a certain fixity of form. Although the curves taken from no two subjects are alike, yet by experience we may learn the limits of variation which are compatible with health.

The physiological auricular complex. *P*, the auricular representative, is found as a summit* in all normal and young adult subjects and in all leads ; it is small and rarely exceeds 1·7 scale divisions in amplitude ; it is followed by a short line which is horizontal or dipping. Amongst the variations of form, which *P* presents in the normal subject, is occasional bifurcation (Fig. 10). *Auricular complexes of these forms are known from experiment to express the origin of the heart beat at the normal site of impulse formation and the passage of the contraction wave through the whole of the auricular tissue in definite directions.*

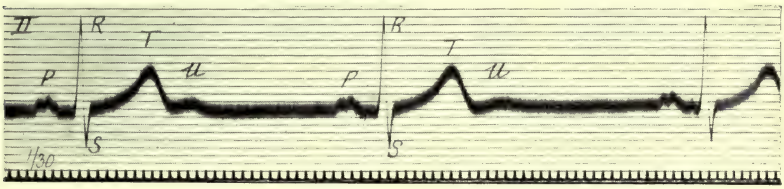


Fig. 10. A normal electrocardiogram, showing a divided *P* summit; a prominent *U* summit occurs at the beginning of diastole.

The physiological ventricular complex. The variations which are found in the type of the physiological ventricular complex are many. *R* and *T* are always present, *Q* and *S* are often absent. All the deflections vary much in amplitude and shape, and *T* may vary in its direction.

The *QRS* group of deflections is of short duration and in normal subjects does not exceed a total duration of one-tenth second. The presence of a *QRS* group of rapid deflections, followed by a slow blunt *T* deflection, is an indication that the ventricular contraction has been propagated from fixed

* *P* and *R* are upward movements when the galvanometric connections are so arranged that if the right arm contact is replaced by the zinc terminal of a copper-zinc couple, and the left leg contact is replaced by the copper terminal, an upward deflection results.

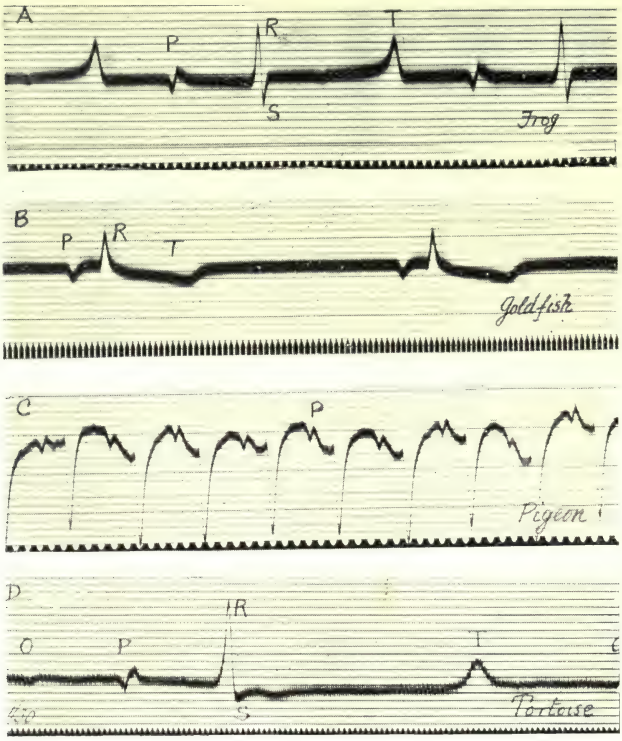


Fig. 11. Electrocardiograms from the several chief classes of lower vertebrata. With the exception of the curves from birds, the remaining electrocardiograms have much in common; the ventricular contraction in the bird seems to originate near the apex.

A. Electrocardiogram from a frog, leading from the middle line, directly above and below the heart.

B. Electrocardiogram from a gold fish; leading from the centre of the left branchial cleft to a point caudal to the apex of the heart.

C. Electrocardiogram from a pigeon; leading from the cephalic and caudal ends of the sternum in the middle line.

D. Electrocardiogram from a tortoise; leading from the cephalic and right hand corner of the pericardium to the left hind limb. Note the sinus deflections at *O*.

points. *It has been awakened by impulses which have traversed the auriculo-ventricular bundle, its main divisions and their arborisations.* This is a conclusion which applies to the

mammalian heart alone, for similar systems of tissue, joining auricle and ventricle, are but imperfectly represented in the lower orders of vertebrata. Curiously enough, however, the electrocardiogram in most of the lower orders is of similar form (Fig. 11), a fact which offers one of the chief difficulties in explaining the individual electrocardiographic deflections.

Certain variations in the form and amplitude of individual deflections require closer description, and it will be necessary to refer to the leads from which the curves are taken. We may confine ourselves to those features which are of clinical importance.

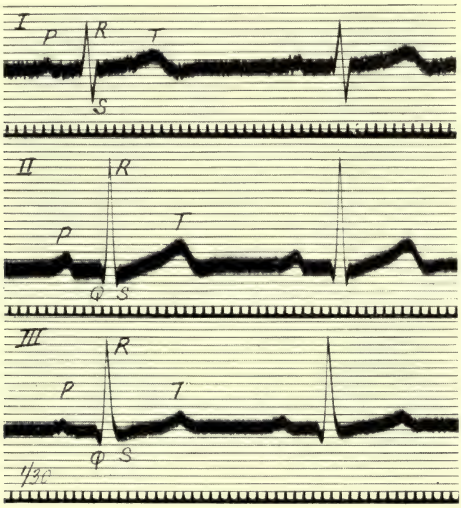


Fig. 12. Electrocardiograms from the three leads in a normal subject. To illustrate the change in the type of curve with change of lead. Note that *R* is tallest in lead *II*.

Curves from the three leads of a normal subject are shown in Fig. 12. It will be noticed that in all *R* is prominent, but that it is most prominent in lead *II*, and that *T* is directed

upwards. On the other hand *Q* and *S* are inconstant. This series of curves may be regarded as of average type. In a series of over 50 young adult subjects the greatest variations presented by serial leads are depicted in Fig. 13 and 14. The importance of these extreme electrocardiograms will be better appreciated when we deal with pathological curves in the next chapter. At present it should be noticed that in Fig. 13 *R* is inconspicuous in lead *I* and most prominent in lead *III*, while *S* is most conspicuous in lead *I*. Precisely the reverse relations are shown in Fig. 14, where *R* is most prominent in lead *I* and inconspicuous in lead *III*, while *S* is deepest in lead *III*. These variations in amplitude are probably the result of relative preponderance of the musculature in right (Fig. 13) and left (Fig. 14) ventricles respectively, and are examples of the pictures which may be found in apparently healthy subjects.

Normal electrocardiograms occasionally exhibit notching of *R* or *S* (Fig. 14); and in lead *III* bizarre types of initial deflections (the *QRS* group) are not uncommon; examples are shown in Fig. 15.

T is always upright in healthy individuals in lead *II*, but it is often inverted in lead *III* and may show partial inversion in lead *I*. Inversion in lead *III* is generally associated with the bizarre *QRS* group to which reference has been made already (Fig. 15), but may occur with the usual form of initial deflections (Fig. 13).

An additional deflection *U* is not uncommon and may be prominent (Fig. 10). This deflection is related to the early events of diastole.

Standard curves from the same subject are almost identical in form and amplitude from time to time. The constancy is such, and the variations in distinct subjects are so definite, that electrocardiograms might well be utilised in the identification of the individual. Considerable changes

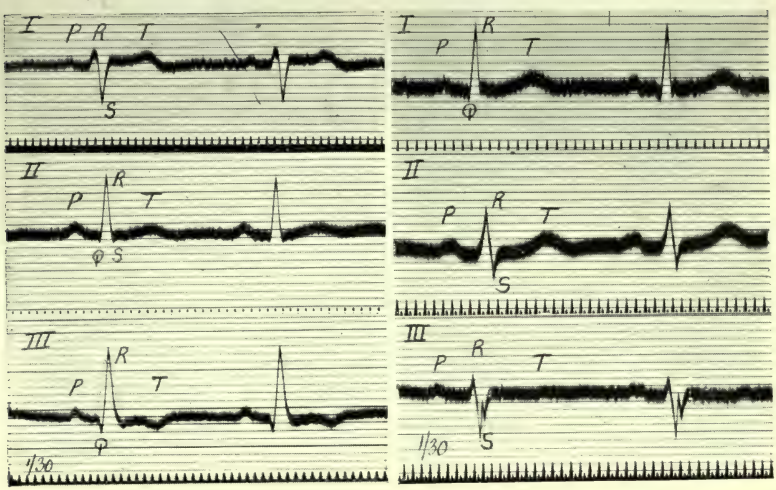


Fig. 13. Normal electrocardiograms showing the tallest *R* in lead *III* and the shortest *R* and deepest *S* in lead *I*. For comparison with Fig. 16.

Fig. 14. Normal electrocardiograms, showing the tallest *R* in lead *I* and the shortest *R* and deepest *S* in lead *III*. For comparison with Fig. 19.

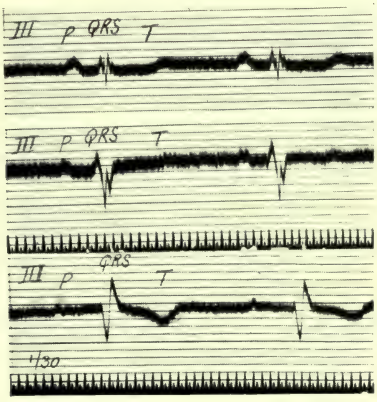


Fig. 15. Normal curves taken from lead *III* in three different subjects. Illustrating the curious arrangements of the initial ventricular deflections which sometimes occur in this lead. They are often associated with inversion of *T*.

in the heights or directions of deflections in electrocardiograms, taken from time to time and under similar circumstances, do not occur in healthy or diseased subjects. An observed change is significant of altered function.

A certain familiarity with the limits of variation in the amplitude of the summits is very essential; it is gained by experience. Some idea of their importance may be found if a series of curves taken from a number of active students are studied. There were in all 59 individuals from whom curves were taken. The limits of amplitude for the several peaks are given in the accompanying table, which includes measurements from 52 subjects. The measurements from seven subjects are

	P	*Q*	*R*	*S*	*T*	*U*
			Lead I.			
Minimum	Trace	0	1·5	0	—0·5	0
Average	0·52	0·51	5·16	2·06	1·93	0·10
Maximum	1	2·0	12·0	6·0	5·5	Trace
			Lead II.			
Minimum	Trace	0	4·0	0	Trace	0
Average	1·16	0·73	10·32	2·23	2·46	0·16
Maximum	1·7	2·5	16·5	4·5	5·0	0·8
			Lead III.			
Minimum	Trace	0	2·0	0	—2·0	0
Average	0·81	0·86	6·61	1·73	0·61	0·06
Maximum	1·5	2·5	14·0	4·0	3·0	0·3

not given; they were rejected from the normal series for various reasons. Six manifested abnormal signs upon the ordinary physical examination, and, of these, *four* gave electrocardiographic curves which showed considerable divergence from those of the selected series. Only one student in whom no other physical signs were obtainable presented divergent electrocardiograms. Two subjects who gave normal electrocardiograms, were found to have, the one a tricuspid murmur,

the other a slight extension of the left limit of heart dulness. These facts speak for themselves. If in any subject electrocardiograms which show considerable divergence from what is regarded as normal are obtained, it is probable that the heart is abnormal. The converse proposition, that if the summits fall within the normal limits of amplitude the heart is probably normal, is also applicable, though not so universally. Hearts which are gravely affected rarely yield normal electrocardiograms. Where degeneration of the myocardium is present, abnormal electrocardiograms may be anticipated.

Electrocardiographic curves are modified by *age* ; the chief change is a decrease in the amplitude of *T* as the subject becomes older. They are also influenced by displacement of the heart. They are modified by *exercise*, notably in the direction of an increase in the amplitude of *T*. But these factors materially influence the interpretation of pathological curves in exceptional cases only.

CHAPTER III.

RHYTHMIC BUT ANOMALOUS ELECTRO CARDIOGRAMS.

Relative predominance of right or left ventricular muscle.

Examination of hearts after death has told us that when the ventricular muscle hypertrophies, it does not do so as a rule in a uniform manner; a relative predominance of the contractile tissue develops upon right or left side. Hypertrophies of the right and left ventricles are conditions of which our knowledge is still imperfect. While purely mechanical influences appear to modify the distribution of the muscle mass as a whole, yet hypertrophy often exists where there is no reason to suspect antecedent alterations of the fluid pressures within the heart chambers, or where the degree of hypertrophy in a particular chamber is insufficiently accounted for by the increased burden which may be supposed to have been born by that chamber. Thus, there is often notable discrepancy between the amount of left ventricular hypertrophy and the grade of damage to the aortic valves, a discrepancy which is frequently unexplained by co-existent defects in the mitral valve. Evidently, there are causes of hypertrophy with which we are unacquainted. If, with few post-mortem comparisons, we attempt to correlate hypertrophy of one or other heart chamber with certain types of electrocardiogram, we shall do so with a full sense that our conclusions may need subsequent revision. And this revision

will be the more necessary because the relative hypertrophy of the two ventricles cannot be estimated from a knowledge of the valve lesions alone, and because the ordinary physical signs which pretend to discriminate between hypertrophy of right and left ventricle are very uncertain.

Yet there are certain valve lesions which are almost always accompanied by a preponderating hypertrophy of the right ventricle. Stenosis of the mitral and pulmonary orifices are perhaps as notable as any, and both these lesions

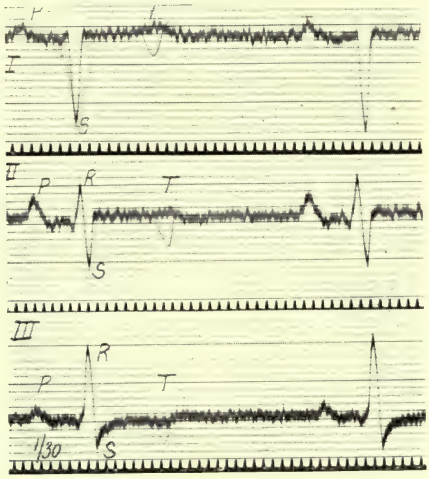


Fig. 16. Curves from the three leads in a case where there was hypertrophy of the right ventricle. Note that there is but a trace of *R* in lead *I* and that *S* is very deep; also that *R* is tallest in lead *III*, while *S* is small. Compare with Fig. 19.

are responsible for very definite physical signs. It is in these hearts that electrocardiographic features, regarded as significant of right ventricular hypertrophy, are almost uniformly present. The features referred to are displayed by Fig. 16. *R* is tallest in lead *III* and shortest in lead *I*, while *S* is deepest in lead *I*. That such divergent electric curves

express right ventricular hypertrophy is rendered extremely probable by a study of the electrocardiograms of the newly born infant. Relative preponderance of the right ventricle during the first few weeks of extrauterine life is physiological; the outlines of the electric curves in the new born child are almost constant; they invariably exhibit the features which have been described (Fig. 17 and 18). The normal adult forms

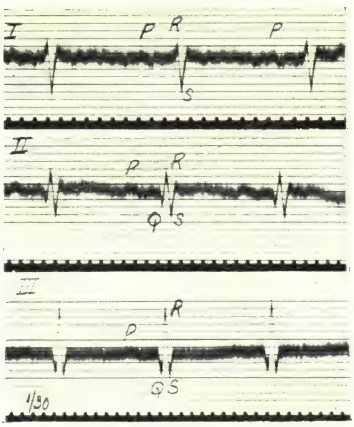


Fig. 17.

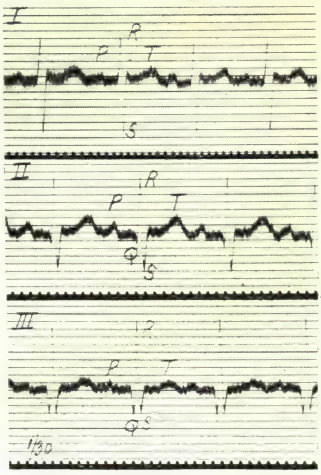


Fig. 18.

Fig. 17. Curves from a child two hours after birth. The relative heights and depths of the peaks is such as is expected where there is relative preponderance of the right ventricular muscle.

Fig. 18. Curves from the same child, but six weeks later. The right-sided preponderance is not evidenced by these curves to nearly the same extent.

of initial deflections are assumed between the ends of the second and third month of extrauterine life. For the present therefore we may regard those curves which show the deepest deflection in lead *I*, and the tallest summit in lead *III* as affording evidence of relative preponderance of the right ventricle.

The most noteworthy conditions in which we expect relative preponderance of the left ventricle are old age, lesions of the aortic valves, heightened blood pressure and renal disease ; but left ventricular hypertrophy is not so commonly uncomplicated as is hypertrophy of the right chamber, and in degree it conforms less to the corresponding mechanical defect. While the electrocardiographic features which are regarded as expressions of left-sided preponderance are generally found in the patients in whom they are expected, notably where there is aortic disease or where there is senile degeneration of the arteries ; yet in a number of patients, the curves speak for left-sided hypertrophy, when no other signs of the condition are found and where the electric signs of this hypertrophy have not been anticipated. And, conversely, other signs of left hypertrophy may seem apparent, or the conditions associated with such enlargement may be manifest, while the electrocardiograms fail to portray it. It is probable that these apparent inconsistencies result in the manners already suggested, and they demand a closer investigation of the phenomena in question. The features of the curves which are considered to portray relative preponderance of the left muscle are the reverse of those seen in Fig. 16. The tallest deflection is found in lead *I*, the deepest in lead *III* (Fig. 19). A prominent *R* in the first lead is

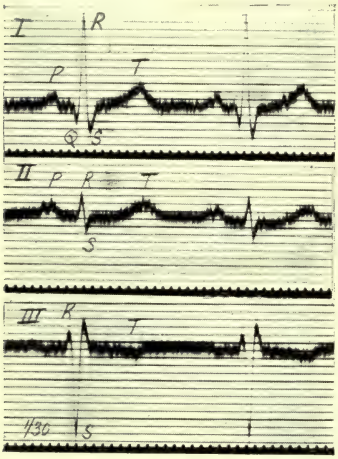


Fig. 19. Curves from the three leads in a case of aortic disease in which the left ventricle seemed much hypertrophied. *R* is tallest in lead *I* and shortest in lead *III*, while *S* is deepest in lead *III*.

accompanied by a diminutive *R* and exaggerated *S* in the third lead.

Inversion of "T."

As the deflection *T* is upright in lead *II* in normal subjects, and is almost always upright in lead *I*; so, inversion of *T* in these leads is always pathological, or nearly always pathological, according to the lead in which it is seen. In lead *III*, inversion has no clinical significance. A good deal of attention has been paid to this deflection in electrocardiograms (see Fig. 24 and 95), and it is a helpful sign in prognosis. Certainly in my own patients, it has often been associated with signs or symptoms of ill omen; and often the patients who have presented it have been short lived. This experience accords with that of other observers.

Aberrant contractions.

In the last chapter, we saw that electric curves are controlled by the direction which the contraction wave takes in the tested muscle. A clear appreciation of this principle and its application is of elemental importance in the interpretation of abnormal electrocardiograms. If a heart beat arises in an abnormal focus, or if the wave of contraction, propagated from the normal focus, deviates from the customary paths, a change in the shape of the electrocardiogram results. Ventricular systoles may be classified, according to the forms of electric curve which they produce.

Now the wave of contraction in the ventricle is propagated from the auricle; the stimulus is conveyed through the auriculo-ventricular bundle, its right and left branches and their arborisations, to the two ventricles. These

structures and their relations to the ventricular cavities are depicted in the accompanying diagram (Fig. 20). If an impulse enters the ventricle through the normal field of reception,

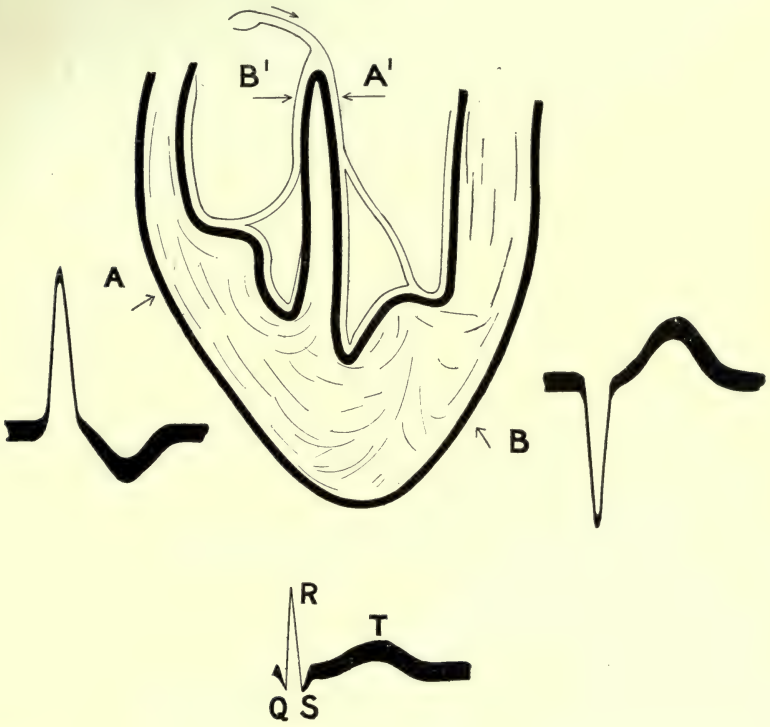


Fig. 20. A diagrammatic view of the ventricles, seen in coronal section. The auriculo-ventricular node and bundle with the main branches and arborisations are depicted. If the ventricle is stimulated through these channels, the type of electrocardiogram is that seen below the diagram. When the heart is stimulated at *A* or the left bundle branch is cut at A^1, a ventricular electrocardiogram of the form seen to the left results. Stimulation at *B* or section at B^1 results in ventricular electrocardiograms of the form seen to the right. The electrocardiograms are those which are obtained from lead *III*.

that is to say, if it travels in the bundle and spreads in a normal manner through the end branches of the bundle, it awakens a normal ventricular beat. It produces a

contraction which displays the familiar electric complex, *Q*, *R*, *S* and *T*. Moreover, these *supraventricular impulses* as I term them are the only ones which generate ventricular contractions of this type.

If the ventricle responds, not to a supraventricular impulse, but to one which reaches it from a single point in its own walls, the electric curve accompanying the contraction is abnormal (see Chapter V on *Premature Contractions*); and whenever this single point varies in position, the electric curve varies also. Each area of the ventricular muscle will give rise, when stimulated, to a different form of contraction; and the differences will be portrayed in the electrocardiogram; but if two points stimulated are close together, the differences in the electric curves will be small or imperceptible. The forms of curve associated with separate areas of the ventricular muscle are known approximately. If a ventricle is stimulated on the right side and towards its base (in the neighbourhood of the arrow marked *A* in Fig. 20), the resultant curve in a vertical lead will be in the main diphasic and of the form depicted to the left in the diagram. It will consist of a steep upward effect, and a slower downward effect. If the area stimulated lies to the left and towards the apex (*B* in Fig. 20) the resultant curve will have the reversed outline, consisting of a steep downward effect and a slower upward effect.

Again, if the impulse is transmitted to the ventricle from the auricle, but the path which it takes is unusual, the curves will be deformed. For example, the impulse travels first through the auriculo-ventricular bundle and arrives at its main division; normally it will pursue its course through both branches simultaneously; but if one branch is damaged, the impulse may pass solely along the other. If the left branch is divided at A^1, then the impulse travels to the right or basal ventricle only; and, although the whole

heart eventually contracts, yet the contraction is abnormal; the electric curve is very similar to that which results when the stimulus is applied in the region of A. Similarly, if the right branch is divided at B^1, the contraction and its electric effect will be very similar to that obtained upon stimulation of the heart in the region of B.

Contractions which are propagated from supraventricular impulses, but which have chosen abnormal channels of passage, I speak of as *aberrant contractions*; and these aberrant

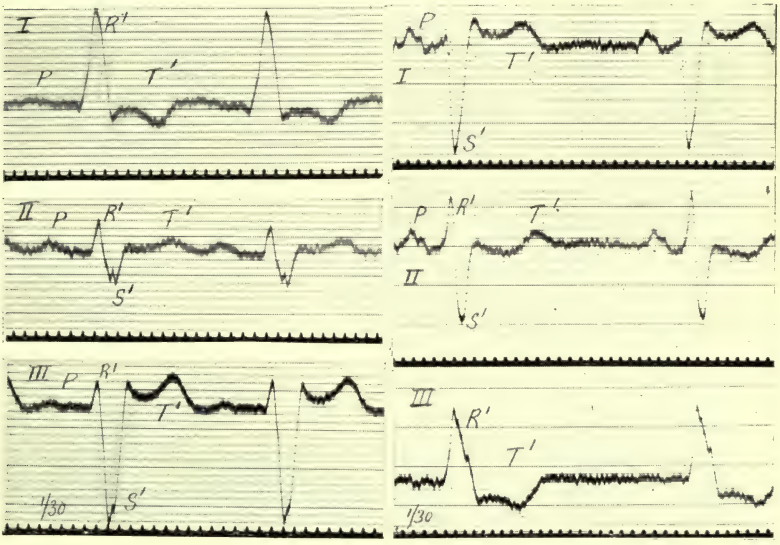


Fig. 21. Curves from the three leads, evidencing functional defect of the right division of the auriculo-ventricular bundle. Note the long duration of the initial phases and the large amplitude in leads *I* and *III*.

Fig. 22. Curves from the three leads, evidencing functional defect of the left division of the auriculo-ventricular bundle. The directions of the deflections are the reverse of those of the last figure, in leads *I* and *III*.

beats are frequently discovered by those who employ electrocardiography as a clinical method. Just as the functions of the main stem of the auriculo-ventricular bundle are frequently defective in the human subject, so

also are those of its individual branches. The forms of electrocardiogram which represent deficiency of the main divisions of bundle are illustrated by Fig. 21 and 22. The auriculo-ventricular sequence of contraction is naturally maintained ; that is to say, each ventricular beat is preceded by an auricular one ; the ventricular complexes are alone abnormal. The chief features of the ventricular complexes are the exaggerated amplitude* of excursion, and the prolongation of their initial phases. The deflections which replace the usual *QRS* group have a total duration which exceeds a tenth of a second, and generally comprise more than a third of the whole complex. When the *right* branch of the bundle is at fault, the abnormality declares itself in the tall and broad summit R^1† of lead *I* and in the deep and broad depression S^1 in lead *III*. Damage of the *left* branch portrays itself in the reversed pictures, a deep broad depression S^1 in lead *I*, and a tall broad summit R^1 in lead *III*. Whether right or left branch is impaired, corresponding deflections in leads *I* and *III* are in opposite directions ; an arrangement which while constant for R^1 and S^1 is generally true of T^1, a deflection which is unusually conspicuous. Lead *II* generally shows deflections similar in direction to those of lead *III*, though they are of lesser amplitude ; but in this there is no uniformity. The two series of curves in our illustrations (Fig. 21 and 22) should be compared with those of Fig. 19 and 16, electrocardiograms from patients with left and right ventricular hypertrophy ; for they present similarities. In the present figures the amplitudes are greater, despite the fact that in these examples they are small for the abnormalities which

* The amplitudes are often much greater than in the curves chosen for illustration.

† The deflections of the curves have no relation to those of the normal electrocardiogram ; I have designated them as R^1, S^1 and T^1 to distinguish them from *R*, *S* and *T*.

they represent ; the opening phases of the ventricular curves are longer by comparison. In Fig. 19 too, T is upright in lead I, while it is inverted in lead III ; whereas in Fig. 21 the reverse is observed. In Fig. 16 and 22, T and T^1 move in the same directions in corresponding leads, but T is relatively inconspicuous throughout Fig. 16. In the curves of lesions

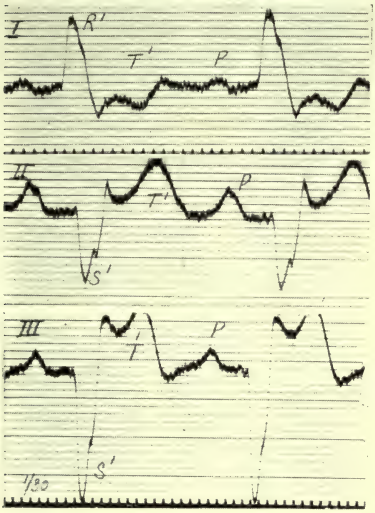


Fig. 23.

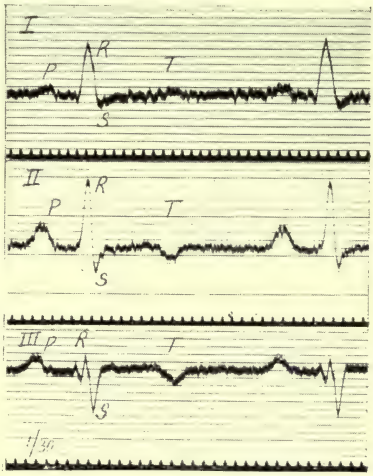


Fig. 24.

Fig. 23. Curves taken from the three leads in a case of aortic disease during a febrile attack. They show defective conduction along the right division of the auriculo-ventricular bundle (see Fig. 21 and 22).

Fig. 24. Curves from the same patient, taken a day later and during the subsidence of the fever. The ventricular portions of the curves have changed profoundly ; there is now no evidence of bundle defect, but of relative preponderance of the left ventricle.

of the right branch, notching of R^1 in lead I, and especially of S^1 in leads II and III is very frequent (Fig. 21 and 23) ; a character which may help a correct analysis.

The points of similarity and contrast between the curves of hypertrophy and bundle branch defects may be emphasised by Fig. 23 and 24. These two series of curves were taken

from a case of aortic disease ; Fig. 23 was taken soon after admission to hospital ; Fig. 24 was obtained a day later. The first figure represents a temporary deficiency of the right branch of the bundle, as evidenced by the amplitude of the curves, the width of the opening phases and the directions of the summits ; the second figure represents the physiological electrocardiograms of this case and speaks for left ventricular hypertrophy, witness the brevity of the opening deflections and the directions of those which are prominent. The directions of T^1 and T in the corresponding leads of the two figures is especially noteworthy ; in each of the corresponding leads, where T^1 points one way, T points the other. The auricular summits alone maintain their forms.

The significance of these electrocardiograms is considerable, and their value can hardly be overstated, seeing that the perversion of functions which they represent can be identified by no other method.* The pathology is similar to that of auriculo-ventricular heart-block, which is frequently coincident. As a permanent condition, damage of a bundle branch speaks for a lesion in this situation, though probably the latter is but a local expression of widespread mischief in more silent areas. The phenomenon is generally accompanied by signs of great enlargement of the heart. As a temporary manifestation, the lesion suggests an acute or subacute process, an invasion of the muscle by an infective agent or poison. Such was the reading in the patient from whom Fig. 23 and 24 were taken. Admitted to hospital, he was found to have an aortic lesion ; the temperature was 100°, but there were no further signs of infection. The electrocardiogram (Fig. 23) showed deficient conduction in the right

* The sole physical sign seems to be conspicuous reduplication of the first sound, which is a frequent though not a constant association.

branch of the bundle, a deficiency which passed away as the fever subsided (Fig. 24). The electrocardiograms provided the sole evidence of an acute myocardial involvement. That the sign is frequent in heart patients is most probable; in how many patients it is overlooked, and how often we are oblivious to the progress of the events of which it speaks, it remains for the future to decide.

As a permanent feature, the right branch is much more frequently defective than the left. Curiously, the former is commonly associated with aortic disease. The death rate amongst patients who exhibit this sign is, in my experience, extraordinarily high.

Chapter IV.

AURICULO-VENTRICULAR HEART-BLOCK, ETC.

Partial heart-block.

The chambers of the heart normally contract in an orderly sequence ; each impulse, generated in the neighbourhood of the sino-auricular node,* courses through both auricles, and arriving at the auriculo-ventricular bundle, the specialised structure which unites auricles and ventricles, is transmitted to the ventricles and these respond to it. There is an appreciable delay in this transmission ; the auricle, as we know, contracts before the ventricle ; and the interval between the onsets of contraction in auricle and ventricle is our index of the length of this transmission time. The electrocardiogram provides one of the most accurate measures of this index which we possess. The interval may be gauged as accurately in the human subject to-day, as in an experiment upon an animal in which auricle and ventricle are laid bare. The measurement is taken from the commencement of P, the auricular summit, to the commencement of the ventricular complex ; as a rule R is used for the sake of uniformity, but Q, which is often the first ventricular deflection, may be adopted if it is thought necessary. The

* A special structure in the mammalian heart and situated at the opening of the superior vena cava. It gives rise to the normal heart rhythm, and in virtue of this function I term it the pacemaker.

P-R interval, as it is termed, varies in normal subjects between ·12 and ·18 seconds in length. Prolongation beyond ·2 seconds is never found in healthy hearts. Such prolongation represents the first stage of heart-block. Fig. 25 illustrates the condition spoken of; in it the *P-R* interval measures ·32 seconds. The sole disturbance, as shown by the

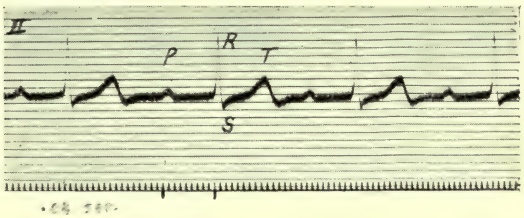


Fig. 25. An electrocardiogram from a case of subacute cystitis, showing prolongation of the *P-R* interval to ·32 seconds.

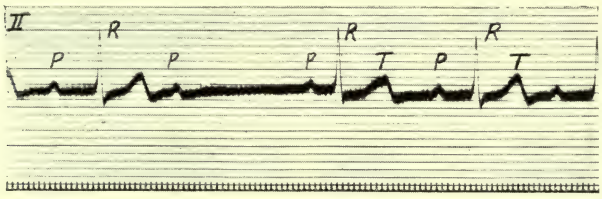


Fig. 26. From the same case, showing a single dropped beat.

electrocardiogram, is an increase of the interval named; individual auricular and ventricular summits are of normal outline. The heart which manifests this change beats regularly.*

The next phase in the progress of heart-block is termed the stage of "dropped beats," where, from time to time, an auricular systole provokes no ventricular response. The electrocardiograms show perfectly regular and normal auricular summits *P*, and most of them are followed by

* rPolongation of the *P-R* interval in patients who are afflicted by epileptic seizures suggests that these attacks are of cardiac origin.

ventricular complexes; but here and there a ventricular complex is missed and the auricular peak stands isolated. This is one of the causes of so-called pulse "intermittence." A simple example of the condition is shown in Fig. 26; measurement will show that the auricular contractions are equidistant; while between the ventricular beats the intervals vary. An interval, of almost double the usual length, separates two of the ventricular contractions; the long cycle is not exactly twice the length of the usual cycle, because, as the figure shows, the *P-R* interval which follows the pause is shorter than the remainder; the ventricular beat which stands at the commencement of the restored rhythm has been moved a little to the left of its natural position, slightly shortening the cycle to the left of it and slightly widening the cycle to the right of it. All the remaining *P-R* intervals are of equal length and are prolonged. These changes in the positions of the ventricular beats are very characteristic of the irregularity. Fig. 28 and 29 exemplify the same phenomena. In Fig. 28 ventricular silences occur after each third or fourth auricular cycle. The analysis of a curve of this kind is readily accomplished: consider the central group of three ventricular beats; the first is preceded by what is evidently an auricular contraction (P^5); it lies in a long pause. A similar auricular contraction (P^9) is found at the end of the next long pause. The remaining auricular contractions are discovered when close attention is paid to the shapes of the *T* summits. T^3, T^4 and T^5 have different forms; the first and last are split; T^4 is tall and pointed. The *T* summits have different forms because *P* summits fall with them. If, in this curve, we take a distance of 6 time-marks from the commencement of an *R* summit and measure to the right, we shall always arrive at the apex of the corresponding *T* summit, for the length of systole is constant in the curve. That the remaining summits are

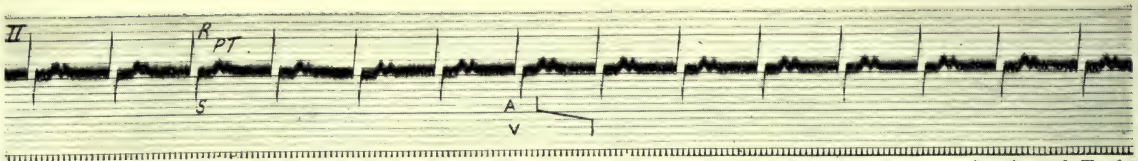


Fig. 27. An electrocardiogram showing prolongation of the P-R interval. P falls before the termination of T of the preceding ventricular cycle. From the same case as Fig. 28.

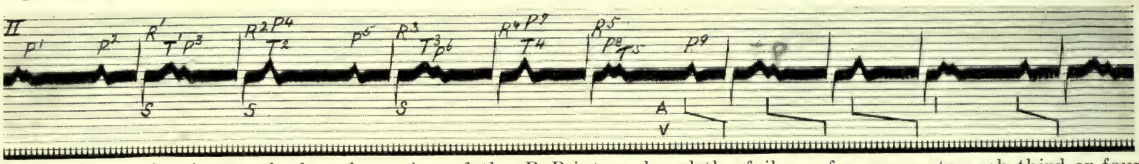


Fig. 28. Curve showing gradual prolongation of the P-R interval and the failure of response to each third or fourth auricular impulse. Partial heart-block of this grade is responsible for some instances of intermittent pulse.

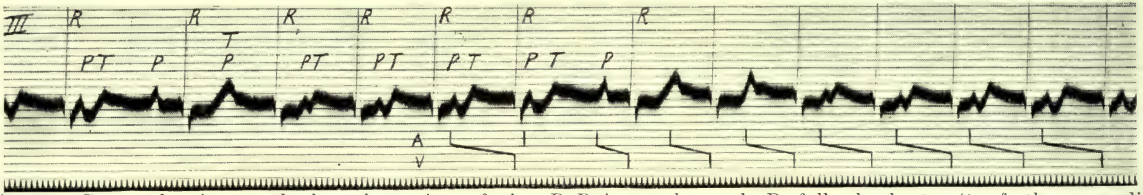


Fig. 29. Curve showing gradual prolongation of the P-R interval, until P falls back to S of the preceding ventricular curve, with failure of response to each sixth or eighth auricular impulse.

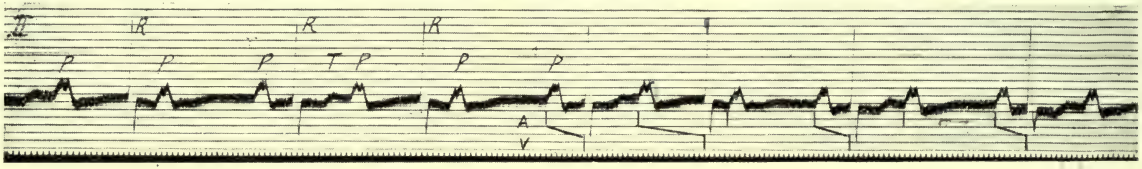


Fig. 30. From a case of mitral stenosis (note the large and bifurcated P summit). Partial heart-block is present, the ratio of auricular to ventricular contractions being at first as 3 : 2, and later as 2 : 1.

auricular is proved by measuring the intervals between them ; P^5 and P^6 are separated by the same interval as P^8 and P^9. The distance between P^6 and P^8 is exactly twice as great, and the intermediate point lies on the summit of T^4. T^4 and P^7 are blended ; the two summits superimpose accurately, whence the tallness of this peak. A perfectly regular series of auricular contractions, therefore, is evident in the curve ; on the other hand, the ventricle beats irregularly. Yet the contractions in upper and lower chamber are related, for the events are repeated. The fifth auricular contraction (P^5) yields the third ventricular response (R^3) ; the succeeding auricular contractions (P^6 and P^7) also stimulate the ventricle (R^4 and R^5), but after progressively greater intervals ; the last auricular contraction of the group (P^8), falling far back into ventricular systole as it does, stands isolated, and there is a ventricular silence before the same events are repeated. The shortening of the P-R interval after the long pause in this figure and in Fig. 26 results from rest and the consequent recovery of the conducting tissues ; following the pause, as impulse after impulse is transmitted, these tissues show greater and greater fatigue, until eventually the stimulus fails to pass. An extremely beautiful example of the same series of events is portrayed in Fig. 29. The analysis proceeds along exactly parallel lines to that just given and is aided by the diagram of auricular and ventricular contractions which has been ruled upon the figure ; but the fatigue comes more slowly and only two ventricular silences are manifest. Fig. 28 has a companion curve, namely Fig. 27. The two curves were taken from the same patient. Fig. 27 was obtained shortly after exercise ; it is noteworthy that all ventricular irregularity was abolished by this exercise, while the auricular rate was somewhat increased. The interpretation of this curve is not evident at first sight ; R and S are readily identified, but T seems double. The first of the two

similar summits represents an auricular contraction in each cycle ; this is known by measurement of the interval between *R* and the summits in this figure and in Fig. 28. The relations are the same as those shown by P^8 and T^5 of the second figure. Thus the *P-R* interval throughout Fig. 27 shows great prolongation ; the auricular contractions do not fall in presystole or even in early diastole, but they coincide with the preceding ventricular systoles. Fig. 30 also shows partial heart-block. Here there is no difficulty in picking out the auricular contractions, for they have the form which they so commonly assume in mitral stenosis. In the earliest part of the figure there is a lack of response to each third auricular beat ; in the last part of the figure what is known as 2 : 1 heart-block, in which alternate auricular impulses alone yield responses, is present. The figure demonstrates the same variation in *P-R* intervals which has been referred to in the discussion of the other figures. A long pause is followed by a short *P-R* interval and a short pause by a long one, according to the preceding period of rest.

Partial heart-block is of very great clinical importance, though it is not within the scope of the present book to discuss it from this standpoint. This has already been attempted in "*Clinical Disorders of the Heart Beat.*" As a permanent condition it tells us of chronic myocardial damage ; as a temporary event, it speaks of an acute heart lesion or poisoning. It will be sufficient if I briefly relate the histories of the curves now presented. Fig. 25 and 26 were taken from a young boy admitted to hospital for subacute cystitis, resulting from an infection by an organism of the *coli* group. While lying in bed for observation he developed an occasional irregularity of the pulse to which little significance was attached. When specially examined, the heart-block was discovered, and it became clear that the heart was invaded, either by the organism itself, or by the

products of its digestion. Appropriate treatment, applied to the bladder, was followed not only by the subsidence of the cystitis, but eventually by restoration of the normal cardiac mechanism.

Fig. 27 and 28 were taken from a young girl who had had an attack of acute rheumatism a year before. The girl came under observation for pains in the neck and chest, and a slight irregularity of the pulse was noticed at her preliminary examination, while she lay quiet. The special examination revealed the disordered heart action which has been described. What was its significance? Partial heart-block appearing in cases of early rheumatic heart disease, is one of our few reliable signs of invasion of the cardiac muscle. Chronic rheumatic heart disease, in its fully developed form, is not made in a day; an unhealthy aortic valve may suddenly collapse it is true, but thickened mitral cusps and hypertrophied and degenerate muscle take years to develop. They develop, to my mind, as a result of repeated infections, often slight in themselves and passing unnoticed. It is along these lines that we may read the curves (Fig. 27 and 28); they are evidences of a slight but acute cardiac muscle lesion, a lesion which is but one step in the course of what may become a chronic and incurable malady. The picture is not a very uncommon one, and often it is combined with a clear source of infection; the channel, maybe the throat, is found and sealed.

The electrocardiogram in this instance took us further; while the patient stood or gently exercised in the recumbent position, the pulse was regular (Fig. 27), and there was no trace of diastolic murmur. Such was the condition when she was first seen. When the pulse was irregular a murmur was audible from time to time. The murmur, so characteristic of early stenosis of the mitral orifice, was heard only when the heart's action was such that the auricular and

ventricular systoles lay separate. When, as in Fig. 28, the contractions were simultaneous and the auricle forced no blood into the ventricle the murmur vanished. These electrocardiograms, therefore, not only explained the nature of the heart irregularity, its reaction to posture and exercise, which was at first so puzzling, but they prompted the search for the characteristic murmur during the irregular heart periods; finally, they told clearly of an acute invasion of the cardiac muscle, in a child, up and about and otherwise seemingly almost well.

The curve shown in Fig. 30 was taken from a case of mitral stenosis in which digitalis was being administered. This drug frequently produces heart-block and that which is shown in the illustration resulted from this poisoning. The electrocardiogram demonstrated that the patient was fully under the influence of the drug.

Complete heart-block.

Partial heart-block is at all times an unstable condition; it passes from one grade to another. In this it contrasts with complete heart-block which is the next disorder to be considered. We have seen that partial heart-block may be present while the ventricular action is regular (Fig. 25), or irregular and while it is rapid (Fig. 27), or slow (end of Fig. 30). Complete heart-block is generally associated with a perfectly regular and slow ventricular action. The auricles beat at their usual rates and regularly, the ventricles beat rhythmically also, usually at a rate of about 30 per minute, and quite independently of the auricles. The electrocardiographic curves portray the condition to perfection and immediately distinguish it from all other forms of slow ventricular action. Fig. 31 is an example; in this curve we see portions of four ventricular beats, placed at regular intervals; each consists of *R*, *S* and *T* deflections of normal

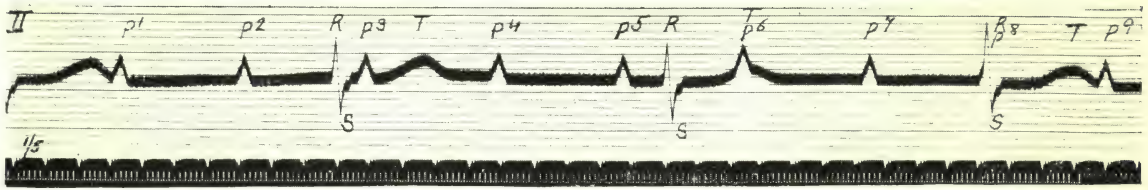


Fig. 31. A curve showing complete heart-block, or dissociation of the auricular and ventricular rhythms. The auricles and the ventricles are beating regularly, but at the independent rates of 78 and 29, respectively. The time marker shows one-fifth and one-thirtieth seconds. The exact superimposition of auricular and ventricular summits should be remarked.

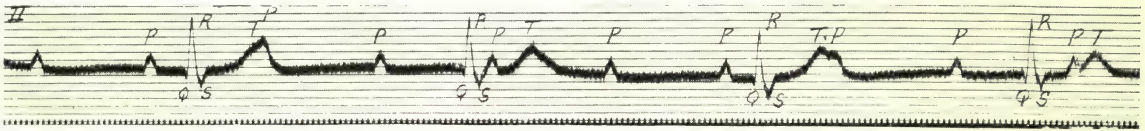


Fig. 32. Showing complete heart-block in another case. The rates of auricle and ventricle are approximately 86 and 35, respectively. Note that each ventricular complex begins with *Q*, showing that the latter belongs to the ventricular systole.

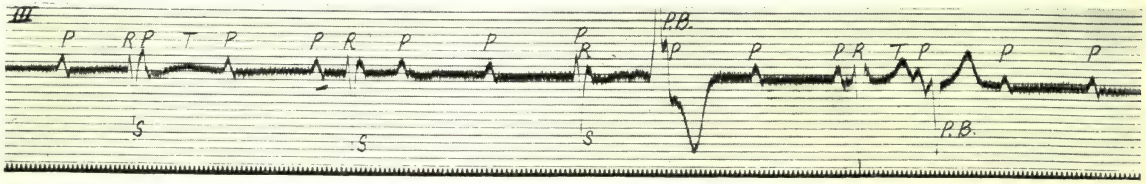


Fig. 33. From a case in which there was considerable enlargement of the heart towards the left side. The curve shows complete heart-block. The regular ventricular action is disturbed by two premature contractions (*P. B.*). The first of these is interpolated.

form. During the period of diastole evident P summits (P^1, P^2, P^4, P^5 and P^7) appear. The remaining P summits fall with the ventricular systoles. One (P^3) falls after S, another (P^6) falls upon T. One (P^8) is obscured by its coincidence with R. They occur equidistantly in the curve. Fig. 28 has already indicated the accuracy with which the electric effects of auricular and ventricular contractions superimpose when these contractions occur simultaneously; it is in complete heart-block that this rule, the constancy of which will be again impressed from time to time, is so strikingly displayed. A number of separate curves have been taken from the same patient and the separate ventricular complexes have been isolated and rearranged one above the other, not in the order in which they occurred, but in the order which renders the superimposition most manifest. The rearrangement is seen in Fig. 34. A single ventricular complex, and two auricular complexes are shown in each curve of this figure. Traced from above downwards, the first auricular summits pass gradually into, through and beyond the opening phases of ventricular systole; the second auricular summits of the curves continue the tale, showing the passage over and clear of the broad summit T. In each instance, where it occurs, superimposition is accurate. The dissociation between the rhythms of the two chambers is thus paraded in the most diagrammatic fashion.

The electric curves of complete heart-block teach us more than that the heart has awakened two distinct rhythms. The slower, or ventricular, rhythm is represented by curves of perfectly normal outline (Fig. 31). They are the result therefore of contractions awakened through the normal channels. The same channels, along which the normal impulse travels from auricle to ventricle, are used by the impulses which create the independent ventricular rhythm. The impulses are of supraventricular origin. Yet these impulses

do not arise in the auricle, for that chamber contracts under the influence of the sino-auricular node. The new ventricular impulses arise in the junctional tissues themselves.

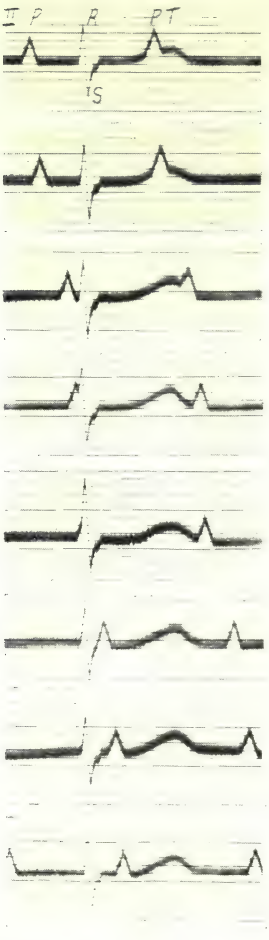


Fig. 34. A figure constructed from the electrocardiograms of the patient, whose curve is shown in Fig. 32. Single ventricular complexes have been re-arranged above each other so as to display the varying relation of the auricular complexes to them.

Fig. 32 is published because it demonstrates that *Q* is a ventricular effect. The curve is again one of complete heart-block, the auricular and ventricular rhythms being independent; but *Q* is constantly associated with the ventricular and not with the auricular contraction; the same fact is clear from an examination of Fig. 29.

Where complete heart-block is present, deviations of the ventricular complexes from the normal outlines are to be interpreted according to the usual rules, as they apply to hearts beating with the normal sequence of chamber contraction. Fig. 33 illustrates the third lead in a case of complete dissociation; the ventricular curves suggest the presence of left-sided hypertrophy. The same figure is complicated by the presence of beats of the ventricle which occur before the anticipated points. They are premature beats (*P. B.*) or extrasystoles, the nature of which will be described more fully in the next chapter. The auricular summits are traceable

throughout the whole curve, whether they fall with the supraventricular or anomalous form of ventricular curve. The curve as a whole serves to illustrate the fact that two forms of disordered action may be exhibited by a heart at the same time.

Slow action of the ventricle.

When partial heart-block is present and only alternate or each third or fourth auricular contraction stimulates the ventricle, and when, too, the auricular rate is normal, then the ventricular action is slow of necessity. The ventricular action is also slow, as has been seen, in the condition spoken of as complete heart-block.

It is very necessary that slow ventricular rhythms of these types should be clearly distinguished from others in which the heart's action is different. To take the most familiar examples first, when the ventricle beats slowly in jaundiced patients or in those who are convalescent from acute infections, the retardation results from a similar slow action of the auricle. The whole heart has a low rate of contraction, but the sequence of chamber beating is maintained. Such slowing is the result of high vagal tone, and the heart beat quickens in response to effort or such drugs as atropine ; the quickening is *gradual* and takes several minutes or more in its accomplishment. To the same class belongs the slow heart action of many healthy adults. All these true brady-cardias exhibit an electrocardiographic picture similar to that shown in Fig. 35 ; each ventricular beat is preceded by a single auricular beat and both are of the normal type.

But there is another and very special form of slow action which involves the whole heart and in which chamber sequence is also undisturbed (Fig. 35). On rare occasions patients are observed, in whom the rate of the ventricle is persistently maintained at between 30 and 40 beats per minute while they

rest. The hearts of which I speak react in a curious way to exercise ; the acceleration is not gradual, there is an abrupt passage of slow into fast rate during the course of a single heart cycle. The curve which is shown was taken from an athlete ; the rate of contraction, while at rest, was usually 36 per minute ; the first effect of exercise was an abrupt rise of rate to approximately double ; further acceleration was gradual ; similar events, though the order of them was reversed, were observed at the cessation of effort. This action of the heart will be spoken of again in a later chapter ; it belongs to a series of phenomena which are at present classed under the term "sino-auricular block." It will be sufficient for the time being to state that it is infrequent, but that its confusion with auriculo-ventricular block should be avoided. The latter is a more serious condition and one which requires greater care in its management.

When the auricular action is slow and the ventricle is starved of its full quotient of impulses, the latter frequently develops the same automatic action as is observed in complete heart-block ; so that although there is no fault in conduction, yet two impulse centres, auricular and ventricular respectively, are active and give rise to responses when opportunity offers. The auricular action usually persists throughout, but if its rate falls below a certain point, or if the automatic rate of impulse formation in the ventricle rises above the same point, then the last named chamber beats spontaneously. Generally this event is recognisable at once in electrocardiograms and an example is shown in Fig. 36. In the first four cycles of this figure, the whole heart is beating sequentially and slowly at a rate of 50 per minute. The fifth cycle shows a ventricular complex of the usual form, but the beat is not a response to an auricular contraction, the *P-R* interval is curtailed and there is partial coincidence of the contractions in the two chambers. This coincidence is still

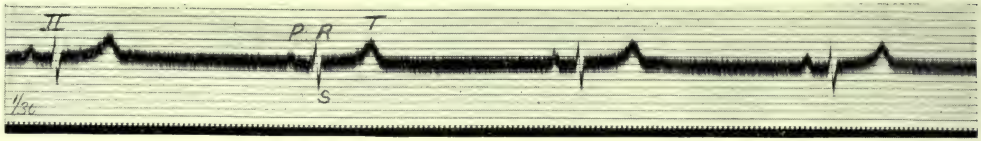


Fig. 35. An electrocardiogram taken from an athlete while at rest, and showing a regular action of the whole heart at 36 per minute. With moderate exercise the rate rose suddenly to double the original rate. The slowing was probably the result of sino-auricular block.

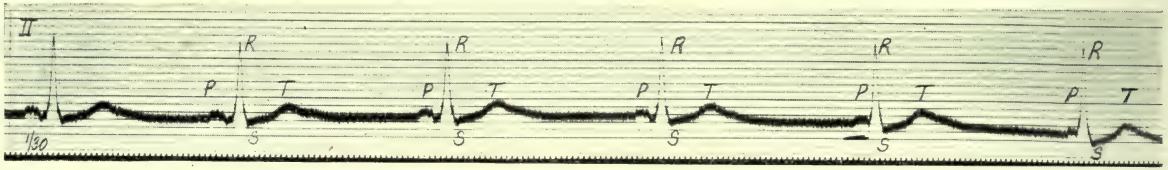


Fig. 36. A curve showing slow action of the whole heart, the rate being 50 per minute, with escape of the ventricle.

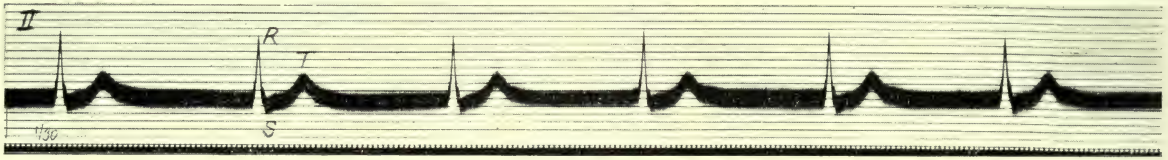


Fig. 37. A curve showing regular and slow action of the ventricle at 48 per minute. No trace of auricular complex is to be found and the line is perfectly smooth in diastole. These curves are usually the result of simultaneous contraction of auricle and ventricle (nodal rhythm).

more pronounced in the last cycle of our illustration, where the *P-R* interval is minute. Now each ventricular complex is of normal outline ; so also is each auricular complex, so far as it is visible ; the ventricular beats are therefore all of supraventricular origin, while the auricular beats are all derived from the natural pacemaker. Yet the ventricular beats are of two kinds ; the first four are of supraventricular origin in virtue of their response to auricle ; the last two are of supraventricular origin in virtue of the impulses formed in the junctional tissues, the usual site of automatic ventricular impulse formation. When the ventricle originates a rhythm of its own it is this centre which is active ; the ventricular beats are termed " escaped " or " automatic " ventricular contractions when they are isolated or in short groups. The bundle is not the only new centre which may come into play when the natural pacemaker is sluggish ; other regions occasionally take up the function of impulse formation and capture the lead ; that centre which produces impulses most quickly is always responsible for the dominant rhythm. It so happens that when the action of the heart is slow, curious electrocardiograms are sometimes encountered. Fig. 37, which exemplifies these remarks, shows ventricular complexes only, and a regular action at 48 per minute. The auricular representatives are not seen. Such electrocardiograms are found when auricle and ventricle contract simultaneously in response to impulses created in the neighbourhood of the auriculo-ventricular node ; the mechanism is consequently termed " nodal rhythm." When the ventricle contracts in response to impulses derived from this centre, its electrical complexes are naturally of the supraventricular type. On the other hand, the auricular complexes will be abnormal, for the auricular contraction wave is propagated in an abnormal direction. Having an abnormal form and falling at the

same time as the ventricular contractions, they cannot as a rule be identified in the curve, which therefore shows ventricular beats only.*

Escaped beats of the ventricle or auriculo-ventricular node, are without clinical significance ; they are simply dependent upon relatively slow action of the natural pacemaker, and it is to this alone that attention should be directed. Yet it is important that they should be recognised when seen, and they are clear in electrocardiograms.

* Another form of " nodal rhythm " is shown in Fig. 63, where there is a simple shortening of P-R interval and an inversion of the auricular complex.

CHAPTER V.

PREMATURE CONTRACTIONS.

Premature contractions, or extrasystoles, are responsible for the majority of those pulse disturbances which are termed intermittences. They are abnormal contractions of the heart, which generally spring from some region of the musculature other than the normal pacemaker or sino-auricular node. I distinguish beats which arise from abnormal foci by the adjective *ectopic*. A premature beat is characterised by appearing before the natural beat, which would continue the regular rhythm of the heart, is due. There are two chief classes of such beats ; those which arise in the ventricle and those which arise in the auricle. Both forms are readily identified in electrocardiograms. A third type arises in the tissues which lie between auricle and ventricle.

Premature contractions of ventricular origin.

When the rhythm of the heart is disturbed by premature ventricular contractions (or ventricular extrasystoles), the disturbance of rhythm is as a rule limited to the ventricle ; the auricle continues to contract at the expected instants. The premature ventricular beats are easily recognised. The electric complexes which represent them are known to be ventricular, because they have the duration of ventricular systoles ; but they occur at earlier instants than those at which ventricular contractions are anticipated ; and they have very anomalous forms. These points are illustrated by

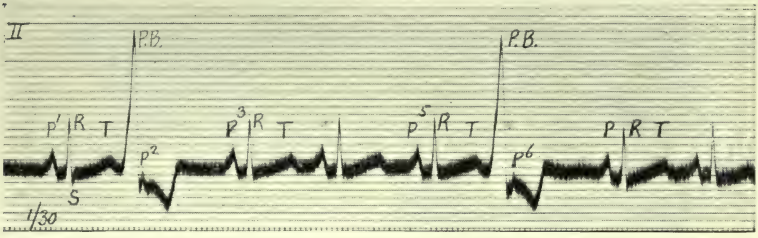


Fig. 38. An electrocardiogram showing two premature contractions, arising in the right and basal portions of the ventricle. The curve shows two buried auricular complexes of normal outline. These premature contractions are responsible for the commonest forms of intermittent pulse (see Fig. 28).

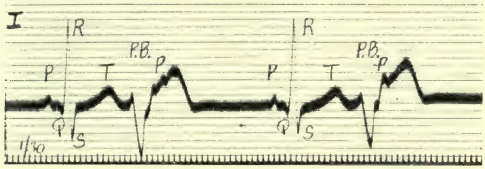


Fig. 39. Premature contractions arising in the ventricle, replacing the alternate normal ventricular beats and giving rise to one form of bigeminy of the ventricle and pulse. The figure illustrates the manner in which buried auricular complexes may be isolated.

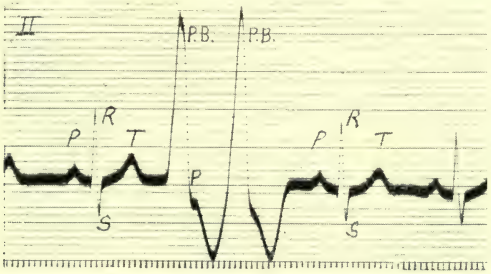


Fig. 40. Two premature contractions arising in the basal or right portions of the ventricle. They occur together and replace a single normal ventricular contraction. The rhythmic auricular contraction falls with the first premature beat.

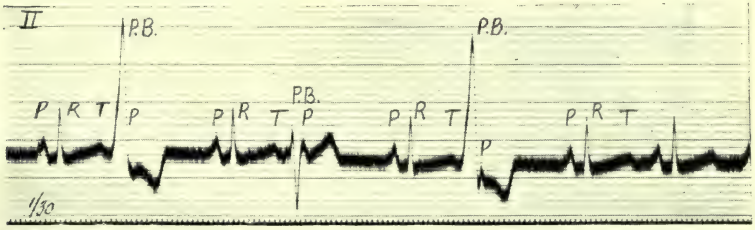


Fig. 41. Bigeminy of the ventricle, resulting from premature contractions of the ventricle. The premature beats arise from two separate foci.

Fig. 38. The figure commences with a perfectly normal heart cycle (*P*, *R* and *T*); this is followed by a tall and pointed summit (*P. B.*) which in turn is followed by a broad downward deflection. The total length of the last complex is the same as the total length of *R* and *T* in the normal cycles. Succeeding the disturbance are three regular cycles and the disturbance is repeated. Now the premature beats of this figure are spontaneous and arise in the ventricle, for no auricular contractions precede them; they are *ectopic*. The auricular rhythm is undisturbed. The distance between P^1 and P^3 is exactly equal to the distance between P^3 and P^5. Where then is P^2? It lies embedded in the complex of the premature beat. That this is so is readily seen by comparing the two premature beats of the figure; when the second occurs, the auricular rate has become a little slower, and consequently the buried *P* summit lies a little more to the right, relative to the premature beat. The two premature beats give complexes of exactly similar forms, the only difference between them is engendered by the superimposition of serial *P* summits which fall with them, and this difference is slight. The recognition of the buried auricular contractions allows the final reconstruction of the whole mechanism. The sequential auricular contractions are now seen throughout; they are placed in regular order. After each premature beat, the ventricle is silent; it is waiting for the next auricular impulse, to which it will respond. The pause which thus compensates for the prematurity of the abnormal ventricular contraction is spoken of as the "compensatory pause." All the auricular complexes of this figure are of normal type, for all have arisen at the pacemaker; all but two of the ventricular complexes are of normal type, for they originate in impulses traversing the normal paths. Two of the ventricular complexes are anomalous in outline, showing that the contraction waves, corresponding

to them, have run abnormal courses; the course has been abnormal because the starting point of the contraction was abnormal.

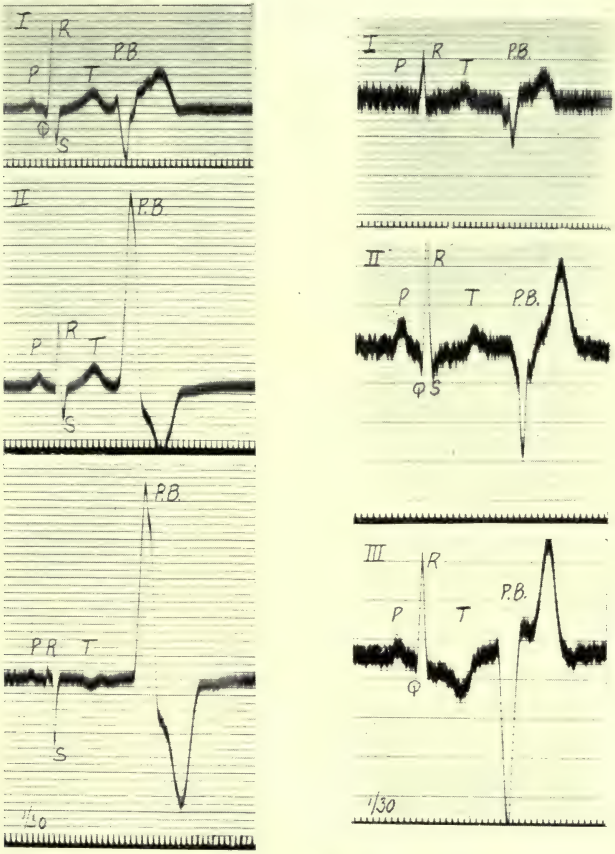


Fig. 42 and 43. Figures illustrating the two chief types of premature contractions of ventricular origin as they are portrayed in the separate leads. Fig. 42 shows a premature beat which arises in the right and basal portions of the ventricle; Fig. 43 shows the corresponding beat from the left and apical portions of the ventricle.

Premature ventricular contractions in different subjects give rise to electric curves of very different forms although the same lead is adopted. The pictures are also different

in different leads in the same individual. Each curve which contains them has consequently to receive separate analysis. Another curve is shown (Fig. 39) which illustrates the manner in which buried auricular contractions are discovered. The premature beats show two main deflections, one downward and pointed and the other upward and rounded. The shoulder of the rounded upstroke is modified by a superimposed auricular representative (*P*) in each case, but it falls at slightly different points relative to the two premature beats. Two successive premature beats are shown in Fig. 40. They take the place of a single normal ventricular contraction. The single rhythmic auricular complex, which is buried, falls upon the downstroke of the first premature beat. If the two beats are compared, they will be found to differ chiefly in this respect. Premature beats from separate ventricular foci are shown in Fig. 41. Two are of the same form as those of Fig. 38; the third or central one consists of three deflections, a short summit, a deep and pointed depression and a rounded summit. The auricular contraction, which falls between the last two, is very prominent and unmistakable.

There are two chief types of premature contractions of ventricular origin: one comes from the basal and right portions of the ventricle (Fig. 42); the other from the apical and left portions of this chamber (Fig. 43; see Fig. 46 also). In any given case the shape of the electrical curves is usually similar in leads *II* and *III*, but usually the direction of the deflections is reversed in lead *I* (Fig. 42), though this is not invariably the case (Fig. 43).

Although premature contractions of ventricular origin are generally followed by "compensatory pauses," this only happens when the impulse of the sequential auricular beat occurs while the ventricle is already in contraction. The ventricle is then "refractory" to stimulation. This is the reason why there is no response to buried auricular

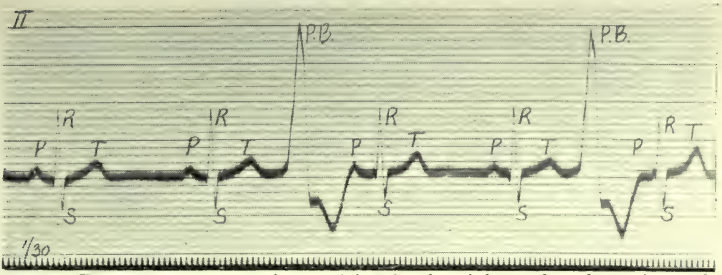


Fig. 44. Premature contractions arising in the right or basal portions of the ventricle and interpolated between normal heart cycles.

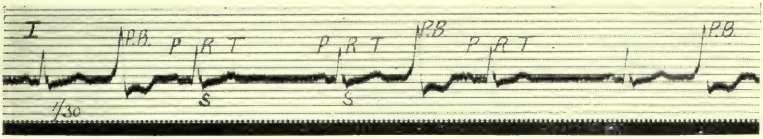


Fig. 45. Interpolated premature contractions of ventricular origin.

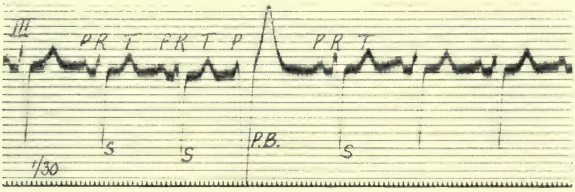


Fig. 46. Premature contraction arising in the left or apical portions of the ventricle.

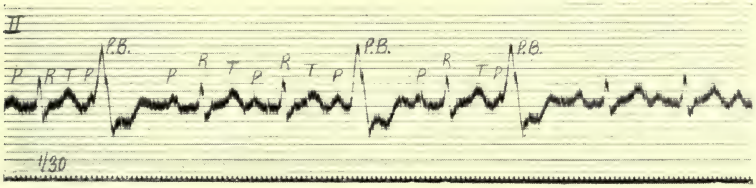


Fig. 47. Premature contractions arising in the ventricle, late in diastole. The auricular contraction has begun, and in one instance is almost complete, before the disturbance occurs.

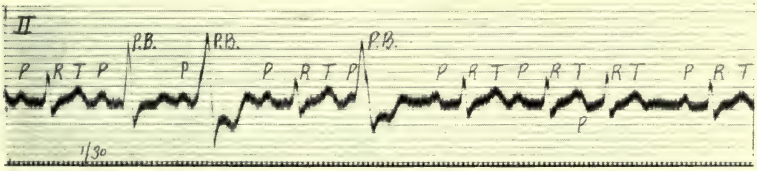


Fig. 48. From the same patient as Fig. 47, showing interference between two waves of contraction in the ventricle (see text). A premature contraction of auricular origin is seen towards the end of this figure.

contractions, such as are seen in Fig. 38 and 39. But if the heart is beating slowly and the premature beats of the ventricle are very early, the ventricular beat may have terminated before the next auricular systole sends its impulse downwards. Under such circumstances the premature beat does not replace a normal ventricular event (Fig. 44 and 45) but is an added phenomenon. Such beats are termed "interpolated."

When a premature contraction falls very late in diastole, the disturbance of ventricular rhythm is slight, for it comes at an instant close to that at which a rhythmic beat is expected. The auricle may even contract before the premature beat appears, so that there is an appreciable, though shortened, interval between the auricular beat and the premature one. The origin of the latter is nevertheless clearly shown by the shape of its electric complex. An example of this phenomenon is seen in Fig. 47. But supposing that the premature beat comes so late that an auricular impulse is already well on its way to the ventricle, then two waves of contraction, one from the normal source and one from the source of irritation in the ventricle, may travel over that chamber and meet somewhere in its walls. Under these circumstances, the electric complex of the ventricular contraction will be of transitional form. Fig. 47 and 48 were taken from the same patient. The usual form of anomalous beats is seen in Fig. 47. Each of the three premature contractions of this figure produce similar electric curves, and each falls after the auricular contraction has started. The first premature beat of Fig. 48 is still further delayed; the interval between it and the preceding auricular beat is just perceptibly less than the normal *P-R* interval. The resultant ventricular curve has a distinct form, in which traces of the normal and traces of the abnormal electric curves are seen. Such a contraction is the result of two impulses giving rise to simultaneous contraction waves, which meet in the ventricular walls.

Premature contractions of auricular origin.

When a new impulse is produced in the auricle, the disturbance is never confined to this chamber. It is usually followed by a responding and premature beat of the ventricle. A simple example of the auricular extrasystole is shown in Fig. 49. The first and second cycles of the figure are normal,

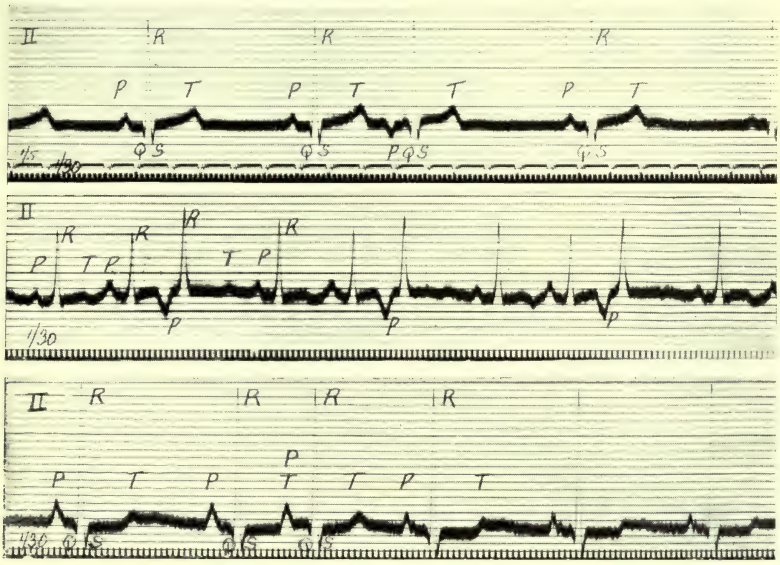


Fig. 49. A single premature contraction of auricular origin. The auricular complex is alone abnormal.

Fig. 50. Three isolated premature contractions, arising in the auricle. The corresponding auricular complexes are abnormal, and there is also slight distortion of the corresponding ventricular complexes (aberration).

Fig. 51. A single premature contraction, probably arising in the immediate neighbourhood of the sino-auricular node.

consisting of *P*, *Q*, *R*, *S* and *T* deflections. The third cycle is premature. Consider first the ventricular complex of this premature beat. It has a perfectly normal outline. The impulse which has given rise to it has consequently travelled along the normal channels ; that is to say, it has come down through the auriculo-ventricular bundle and its branches.

It is a ventricular beat of supraventricular type (see page 30). When we search for the cause of this ventricular beat, we find it in the auricular contraction which has immediately preceded it. But this auricular beat is of abnormal type, the electric curve shows a depression and not a summit. To what is the

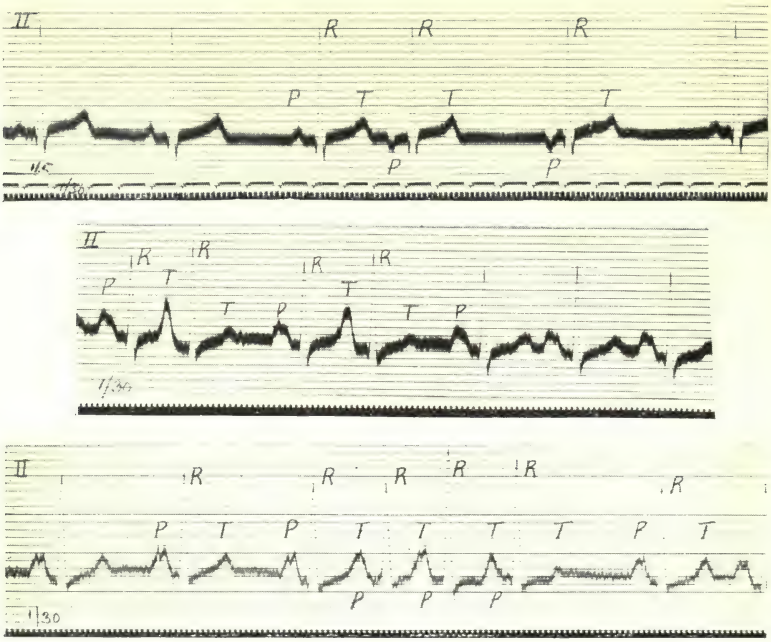


Fig. 52. A single premature contraction of auricular origin. The first beat after the pause originates in the same point as the premature one.

Fig. 53. Two isolated premature contractions of auricular origin. The premature auricular systole coincides with the preceding ventricular contraction ; *P* and *T* are thus superimposed. Taken from a case of mitral stenosis.

Fig. 54. Three successive premature contractions of auricular origin. Taken from a case of mitral stenosis ; note the split *P* summits in this and the preceding figure.

inversion due ? It is the result of the abnormal path which the wave of contraction has taken in the auricle. The impulse, from which the whole premature heart cycle results, has been generated in an abnormal auricular focus, *i.e.*, in a

region lying at a distance from the pacemaker. It has arisen from an *ectopic* centre of impulse formation ; the wave of contraction has therefore passed through the auricle in a direction other than normal ; but as it arises in the auricle, it needs must travel along the only path to the ventricle, and this is constituted by the normal channels. Usually, the pause which follows a premature auricular contraction is not compensatory, it is too short, and consequently there is a disturbance of the fundamental rhythm of the heart. A rare example of premature auricular contractions, in which the pause is almost, if not quite, compensatory, is shown in Fig. 50. The auricular complexes are inverted in this figure, as they are in Fig. 49. When a premature beat arises in or near the sino-auricular node, then the whole premature electric curve is of normal form, as shown in Fig. 51 ; the *P* summits are similar throughout, showing the similar origins of the beats ; moreover, the pause which follows the premature beat is unduly shortened ; its length is either the same or somewhat shorter than that which follows the usual rhythmic beats.

On all but rare occasions, the beats which follow a premature contraction are derived from the pacemaker ; the old heart rhythm is immediately restored (Fig. 49) ; but it sometimes happens that the first beat of the returning rhythm is also ectopic (Fig. 52) and springs from the same centre as the extrasystole. A not dissimilar disturbance, but of higher grade, is shown in Fig. 63.

When new impulses are created in the auricle, the premature auricular contraction may fall so early as to coincide with the preceding ventricular systole. *P* and *T* then fall together and superimpose. Superimposition of these electric summits is seen in Fig. 50 and 51, but it is clearer in Fig. 53, because the *P* and *T* summits are prominent. The second and fourth beats of the last named figure are premature and arise at an ectopic auricular focus. The *P*

summit of the premature beat coincides with the T summit of the preceding cycle and combines with it to form a tall blunt summit. That the premature beats are in reality ectopic is known after a careful consideration of the shapes of the summits in question. Simple superimposition of P and T would not yield a united summit of the type depicted, for the rhythmic P shows a bifurcation in this case.

An example of premature auricular contractions, occurring in succession, is shown in Fig. 54. Each premature auricular complex coincides with a preceding T summit. All told, there are three of them.

While it is the general rule that the ventricular complex of a premature auricular contraction is of normal outline, yet divergence from this type is often encountered, and is sometimes so great that unless it is recognised, mistakes in interpretation are apt to occur. The premature R summits of Fig. 50 and 53 are taller than those of rhythmic beats; but the difference in general outline is but slight. Examples of the chief forms of conspicuous divergence are illustrated in Fig. 55, which consists of four strips of curve taken from the same patient. The first curve shows a premature auricular contraction which conforms to the general rule; all the ventricular complexes of this strip are similar; the premature beat (the third in the curve) is evidently of supraventricular origin. The abnormal auricular complex notches the upstroke of the preceding T summit; this is appreciated when T^1 and T^2 are compared; they have not the same shape, and the difference is due to an inverted P falling with T^2. The second and third curves show precisely similar events, T^3 is notched by an abnormal auricular representative in both instances; but in these curves the premature auricular contractions give rise to ventricular beats of anomalous type. In the second curve the complex consists of two upward summits, the first of which is split; in the

third curve upward and downward deflections of equal extent are followed by a rounded summit. Both the curious beats were of supraventricular origin ; both resulted from auricular impulses; the abnormality of the ventricular curves is attributed to deficient conduction along certain tracts of the junctional

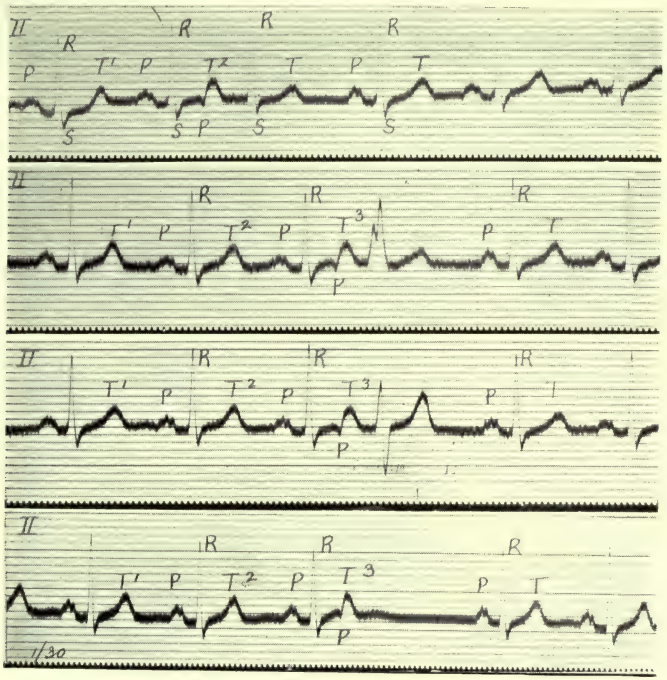


Fig. 55. Four curves from a single subject. Each shows a solitary premature auricular contraction. The premature auricular complex falls with the commencement of the preceding *T* and notches it. The corresponding ventricular complexes of the first three curves are of various forms ; the central curves illustrate a phenomenon which is described under the term " aberration." In the last curve the premature auricular contraction is blocked.

tissues, whereby the auricular impulse has an abnormal distribution in the ventricle ; the deficiency is confined to the premature beats because rest has been brief. The electric curves exemplify a phenomenon which has been

described already under the term aberration (see page 31). They are almost always taken from patients in whom conduction defects are demonstrable. The conduction intervals of the premature beats are prolonged in the second and third curves of this figure ; and in the fourth or last curve a premature auricular contraction is blocked ; T^3 is again notched by an inverted P, but, as opposed to what happens in the other curves, the ventricle fails to respond to this auricular contraction.

Premature contractions arising in the junctional tissues.

Although the majority of premature contractions arise in the auricle or in the ventricle, on rare occasions the conducting tissues, which unite these two chambers, appear to originate the disturbance.

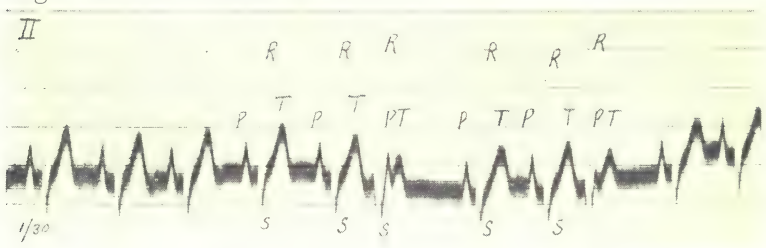


Fig. 56. Two premature contractions arising in the junctional tissues.

There are two premature ventricular systoles in Fig. 56 ; and the relations of the auricular and ventricular contractions in this figure as a whole are such as are found when the premature beats are of ventricular origin. The pauses are compensatory, and there is no disturbance of the auricular rhythm. The auricular contractions are placed at regular distances in the curve ; each is represented by a normal P summit, though two of these summits are buried. The undisturbed auricular rhythm demonstrates that the premature beats have arisen below the auricle. Yet they have

had their origin above the main division of the auriculo-ventricular bundle, for the ventricular complexes have physiological outlines ; the impulses have been supraventricular. The focus of disturbance, in such cases, has been the main stem of the auriculo-ventricular bundle.

Premature contractions, whatever their form, are always very easily recognised in electrocardiograms ; in other graphic records they often produce most confusing pictures, and this is frequently the case when they appear in groups. The characters of the pulse may closely resemble those which are associated with grave disturbances such as auricular fibrillation, heart-block and alternation. Premature contractions are relatively benign, and as electrocardiography is often the only certain method of differentiating this irregularity from more serious disturbances, it is to be recommended as a final means of decision in instances of doubt.

CHAPTER VI.

SIMPLE PAROXYSMAL TACHYCARDIA.

Electrocardiography has explained many of the phenomena of simple paroxysmal tachycardia. In the first place it has shown that these transient attacks of cardiac acceleration are not the result of simple disturbances of innervation as was at one time thought. If a heart accelerates in response to exercise, emotion, fever or other such cause,

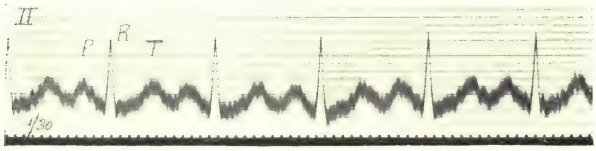


Fig. 57. An electrocardiogram from a case of exophthalmic goitre. The heart rate shown is 150 per minute. The curve indicates a simple acceleration of the normal rhythm.

then the electrocardiogram has in general a physiological outline Auricular and ventricular complexes retain their shapes, except for minor modifications which are the simple result of the increased heart rate. Fig. 57 was taken from a patient who suffered from exophthalmic goitre, the ventricular rate being 150 per minute. The curve shows *P*, *R* and *T* summits ; and these have the conformation which stamps the heart beats as propagated from the natural pacemaker.

The curves of paroxysmal tachycardia are different ; they show that the dominant centre of impulse formation is

displaced during the attacks, though as a rule it is still to be found in the auricle. Fig. 58 and 59, are from a case of paroxysmal tachycardia ; each figure shows the three leads. The first (Fig. 58) was taken while the heart action was natural, its rate being 81 per minute. The shapes of the auricular and ventricular complexes in these curves should be noticed and compared with those of Fig. 59, which depicts a paroxysm of acceleration at 146 per minute. It is seen that the ventricular complexes of the paroxysm retain their form and that they do so very faithfully. Each ventricular

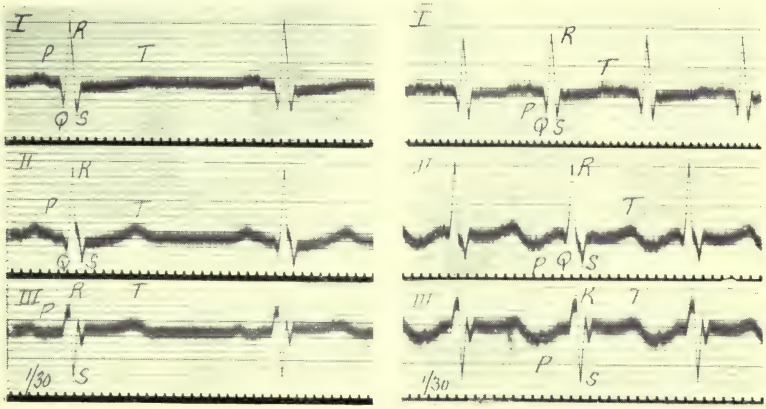


Fig. 58 and 59. Two sets of curves from a case of simple paroxysmal tachycardia. Fig. 59 was taken while the heart beat slowly ; Fig. 60 while it beat rapidly. The curves demonstrate the supraventricular origin of the paroxysm. The inversion of *P* in leads *II* and *III* of Fig. 60 indicates that it arose in an ectopic auricular focus.

summit or depression of Fig. 58 is also found in Fig. 59. The similarity extends even to the minutiae of the curves ; the notch on the downstroke of *R* in lead *II* and the notch on the upstroke of *S* in lead *III* are repeated. The ventricular beats during the paroxysm were therefore of the same kind as those of the slow period ; in both phases the impulses pursued the same course ; that is to say, they travelled along the normal paths

of the junctional tissues. The paroxysm was therefore of supraventricular origin. But when we examine the auricular complexes in the two sets of curves, it is seen that while the *P* summits of the slow period are upright, those of the paroxysmal period are inverted in leads *II* and *III* and are also different in lead *I*. These changes are significant, for they tell us that when the paroxysm comes, the natural pacemaker is no longer the dominant impulse centre, but that some new auricular focus has an enhanced activity and that the old centre is outpaced. It not infrequently happens that such a conclusion as to the nature of the paroxysm can be maintained from its intrinsic beats alone. Yet it is always more justified when the curves of the two distinct phases of heart action can be taken, and when the similarities and dissimilarities which have been named are found.

The similar nature of paroxysms of tachycardia and solitary premature beats is at once suggested by the observation that the abnormal beats are of *ectopic* origin. There is in fact no essential difference between the individual beats of the one and the other. Premature beats are not always solitary ; they sometimes occur in short groups (Fig. 40 and 54) ; it is entirely a question of the length of these groups and the nature of our terminology as to whether we term such small groups successive premature beats or short paroxysms of tachycardia. In one and the same case, isolated beats, short groups and longer paroxysms may be found, and the electric curves usually show that all originate in the same focus (Fig. 60). Premature beats are characterised by the relatively short pauses which precede them ; the rapidity of impulse formation is expressed in the paroxysmal curves by the rate of the heart beat and by the prematurity of the first paroxysmal beat. That the two phenomena are essentially the same is also demonstrated by the manner in

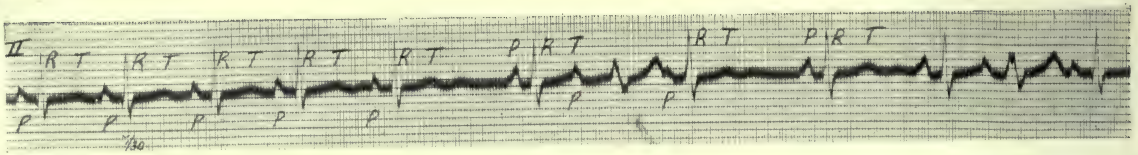


Fig. 60. The end of a paroxysm of tachycardia of auricular origin, and the commencement of the slow normal rhythm, interrupted by premature contractions. The last are auricular and two of them show ventricular complexes of aberrant type. The time marker in this curve rules vertical lines ; a pair of lines occurs at each thirtieth of a second.

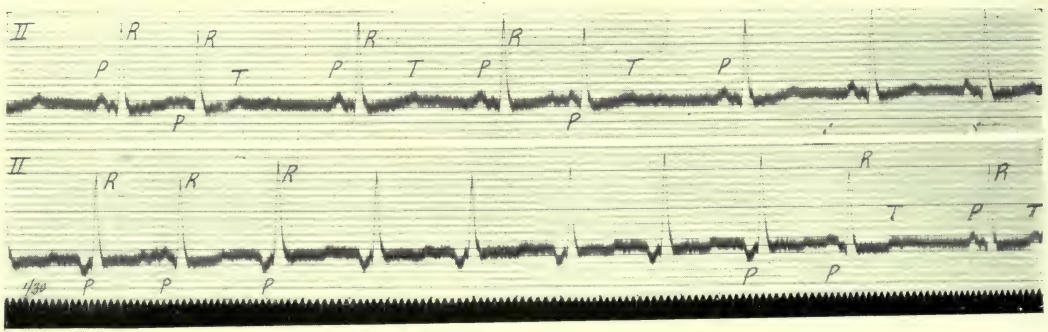


Fig 61. Two curves from a case of simple paroxysmal tachycardia ; the upper one is from the period of slow and the lower is from the end of a period of rapid heart action. The curves show the auricular but ectopic origin of the paroxysm. Both slow and fast rhythms are interrupted by occasional premature beats having a common and auricular focus of origin.

which each subsides. The solitary premature beat, and the paroxysm, are succeeded by a pause which in a single case is of constant length, and there is an immediate return of the normal rhythm (Fig. 60). The change from one rhythm to the other in paroxysmal tachycardia is absolutely abrupt and in this way contrasts with the gradual retardation of a simple cardiac acceleration. Examples of curves which show the offsets of paroxysms of tachycardia are shown in Fig. 60, 61 and 62, and as these curves are published to illustrate special features of paroxysmal tachycardia they may be described in more detail.

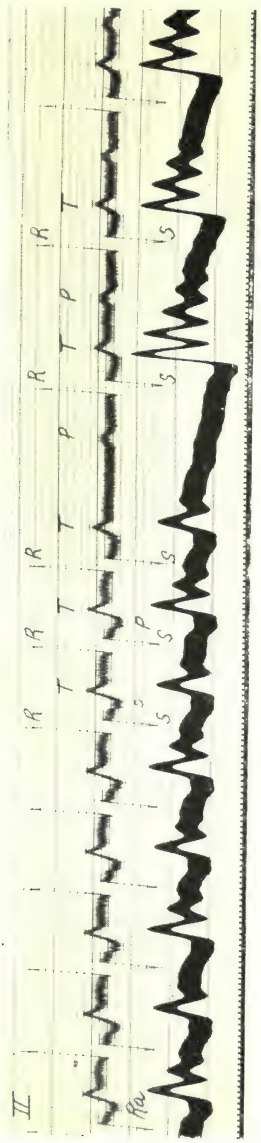


Fig. 62. The end of a simple paroxysm of tachycardia which arose in the auricle. There is delay in conduction during both slow and fast periods; during the latter the auricular contraction falls with the preceding ventricular systole. During the paroxysm the pulse shows alternation. There is also slight alternation in the heights of *R* summits, the *larger R* corresponding to the smaller pulse beat.

In Fig. 60 the first five cycles are paroxysmal; these heart beats arise in a new auricular focus; this is known from a comparison of the five paroxysmal beats with that which immediately succeeds them, the auricular summits are at first steep and small; with the return of the normal mechanism they are larger and of the customary form. The first normal cycle is succeeded by two premature contractions, each of which springs from the

auricle and from the same focus as the paroxysmal beats ; the first premature beat has a ventricular complex of aberrant type (see Fig. 55, second curve, and explanation). A pause follows the two premature beats and it has the same length as the pause following the paroxysm. Then come two normal cycles and after the last, two premature auricular contractions again occur. The figure clearly shows those associations between paroxysms of tachycardia and premature beats which have been mentioned already.

In Fig. 61 are two curves from the same case. The first exhibits a normal rhythm disturbed by two premature auricular beats. The *P* summits which are premature are much smaller than those of the rhythmic cycles. The second curve shows a paroxysm of tachycardia in the same patient ; the ventricular complexes are unaltered but the auricular ones are inverted. The second and last cycles of this paroxysm are premature, they are derived from the same source as those which disturb the normal rhythm in the first curve. A longer pause terminates the paroxysm and the first beat of the returning normal rhythm is shown. Thus, in this patient, paroxysms of tachycardia, thrown in from a second auricular focus, disturb the normal rhythm ; and both the paroxysms and the normal rhythm are interrupted by single premature contractions from a third focus.

Fig. 62 exhibits an electrocardiogram and radial pulse curve and contains the end of a paroxysm of tachycardia and the beginning of the slow rhythm. The post-paroxysmal pause is well displayed and there is a little quickening of the heart rate at the beginning of the slow rhythm, a common phenomenon. If the ventricular complexes of the slow and fast periods are compared, they will be found to be alike, except that at its commencement *T* goes deeper during the paroxysmal stage. This change in the shape of *T*, which is semi-inverted while the heart rate is fast, is not incompatible

with acceleration alone. Had we simply the paroxysmal curves, or the curves of the slow and fast periods, then a complete analysis of the paroxysmal mechanism would not be feasible. The record of the transition from one form of heart action to the other provides us with the full data. If the last two cycles of the paroxysm are compared, it will be clear that T alters its shape ; the semi-inversion is not present in the last paroxysmal cycle ; it is due, in the other paroxysmal beats, to the superimposition of an inverted P deflection upon T. The paroxysm has had its origin in a new auricular focus and each auricular systole has propagated a ventricular systole ; the rate is fast (128 per minute) and the P-R interval is long (·33 seconds) ; and so each auricular contraction falls with the preceding ventricular contraction. The last ventricular beat is necessarily uncomplicated in this respect, for the auricular acceleration has terminated before this systole appears. The paroxysm ends in the usual post-paroxysmal pause and the natural pacemaker re-establishes its control ; but the prolongation of the P-R interval is maintained.

The curves used as illustrations fully emphasise the value of comparing the electrocardiograms of fast and slow periods. The curves of the paroxysmal phases are but rarely sufficient to provide a full analysis. The curves of both rapid and slow heart action may also be insufficient ; the curves which show the transition from fast to slow or from slow to fast are the most valuable of all.

Paroxysms of tachycardia may also arise in one or other ventricle ; under these circumstances the electric curves of individual beats are of the forms seen in Fig. 42 and 43. But tachycardia of ventricular origin is a rare phenomenon. An auricular origin is the general rule. It is probably because the ventricular disturbances are less compatible with life that they are so rarely recorded.

The chief value of electrocardiographic records in tachycardia is in distinguishing between simple acceleration, simple paroxysmal tachycardia and a third condition which is described in the next chapter. The natural history of the three conditions and their reactions to treatment are essentially different. Rapid heart action of obscure origin is frequent; the electric method singles out those which are primarily of cardiac origin and distinguishes them from simple disturbances of innervation.

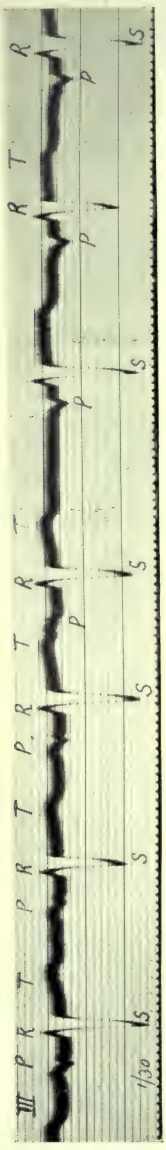


Fig. 63. From a patient who presented hypertrophy of the left ventricle. A regular rhythm of sinus origin is disturbed by a single premature beat arising in the auricle. Following upon the pause a rhythm of ectopic auricular origin is shown; there is some shortening of the *P-R* interval in these last three cycles; the new rhythm probably arose from the lowest level of the auricle, namely the auriculo-ventricular node.

Dislocation of the pacemaker.

It may be profitable to contrast simple paroxysmal tachycardia with the disturbance of the heart's action which is shown in Fig. 63. This electrocardiogram opens with three cycles in which the heart beats have been propagated from the natural pacemaker, as is evidenced by the shape of the complexes (*S* is deep because the curve was taken from a patient in whom there were evidences of left-sided hypertrophy; the curve is from lead *III*). The fourth beat is a premature contraction of auricular origin. The usual pause succeeds this disturbance, and subsequently rhythmic heart action is resumed. But the restored rhythm arises from a new auricular

focus, as is shown by the shape of the auricular complexes. There is little or no difference in the rate of the heart before and after the disturbance, and in this respect the curve contrasts with those of paroxysmal tachycardia. The ectopic rhythm is developed in this instance from the same type of impulses as is the normal rhythm ; unlike the paroxysms of rapid heart action, it has no affinity with premature beats in the manner of its production.

CHAPTER VII.

AURICULAR FLUTTER.

The term "auricular flutter" is used in this chapter to designate extreme grades of auricular acceleration. I employ it when, during periods of acceleration, the auricular rate is maintained above 200 per minute. The disorder is one which in the untreated state can rarely be diagnosed by any method other than the electrocardiographic. The distinction between "flutter" and the condition described in the last chapter under the term "simple paroxysmal tachycardia" may ultimately prove too arbitrary; nevertheless it has present advantages, for although the two conditions so separated have a number of features in common, yet in certain ways they do not behave alike. From the purely clinical standpoint their distinction at the present time is of decided importance.

The usual rate of auricular contraction in flutter is approximately 300 per minute; it may reach 330 per minute. The flutter comes abruptly and goes abruptly, as does a simple paroxysm of tachycardia; the two conditions have this common characteristic and almost certainly possess a common pathological basis. Flutter also occurs in short paroxysms, but it tends to persist for periods of time which are measured in months or years. In this respect "flutter" has an affinity with another mechanism, namely "auricular fibrillation," which is described in the next chapter.

The auricular rate is so rapid that the ventricle is rarely able to follow it ; and so it happens that most patients who exhibit flutter, also demonstrate heart-block, the grade of which is generally such that alternate auricular impulses stimulate the ventricle. Most patients who are the subject of flutter have an enhanced ventricular rate, but the ventricular rate is but one half the auricular. On the other hand, the ventricular rate may be slow ; any grade of block may be present.

The electrocardiograms.

The electrocardiograms are often puzzling or actually misleading and always require careful analysis. It is often of

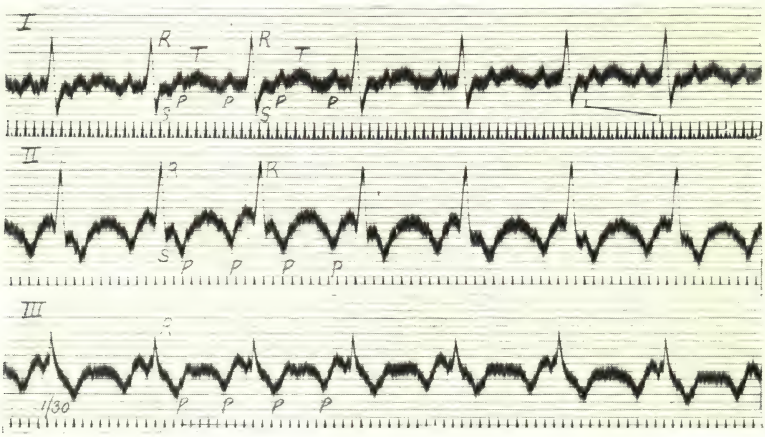


Fig. 64. Curves from the three leads in a case of auricular flutter. The auricular rate is 324, the ventricular rate is 162 per minute. In leads *II* and *III* the auricular complexes are contiguous.

advantage to have the curves from the three leads side by side, so that the same or different events may be compared or contrasted in distinct leads. Fig. 64, which contains all three leads, may be used to illustrate the chief points in the analysis. In lead *II* of this figure the most conspicuous peak is *R* ; it is followed by a small depression *S*. These are the only certain indications of ventricular systole. The remainder of

the curve might be described as consisting of two V shaped depressions, one of which seems to correspond to an inverted *T*, the other to an inverted *P*. The two depressions have an almost exactly similar shape. Now this is not inconsistent with the interpretation, which at first seems warranted, that we have to deal with an inverted *P* and *T*, for similarity of the two deflections is not uncommon. But it would be a strange coincidence if, with this similarity, *P* and *T* should be placed at exactly equal distances from each other throughout the whole curve. Moreover, even though *P* and *T* may resemble each other in a given lead, *they never do in the three separate leads* ; yet in each of the curves of our illustration a uniform series of waves is present. In lead *III* they are of much the same form as in lead *II* ; while in lead *I* they appear as small upwardly directed and pointed summits. In all leads they lie at equal distances from adjacent summits of the same form ; so that in all leads the deflections have a perfectly uniform incidence. These facts demonstrate that the two waves have a similar origin ; and, although one lies in ventricular diastole, that they are the result of auricular systoles. Fig. 64 therefore shows 2 : 1 heart-block, the rate of the auricle being 324 and that of the ventricle 162. In leads *II* and *III* the real *T* is scarcely distinguishable, but in lead *I* it is clear, having a broad form and falling between two *P* summits. The ventricle is responding to alternate auricular impulses ; but is the response to the first or the second auricular contraction ? To answer this question we have first to determine the points upon the curves which represent the onsets of auricular systoles. This may be accomplished in lead *I* in which the upstroke of *P* is relatively steep ; and this upstroke is found upon measurement to correspond to the upstroke in leads *II* and *III*. The auricular representative commences in an upward deflection in all the leads of this figure. The response of the ventricle is not to

the auricular contraction which immediately precedes *R*, but to that which falls with *T*, for the first interval is too short.

The electric representatives of auricular contractions, when this chamber is in a state of flutter, are usually contiguous. In Fig. 64 this is best appreciated in lead *II*. *P* commences in an upstroke but is continued in a dome like summit which ends at the foot of the succeeding upstroke. As one upstroke precedes *R*, so the dome into which it

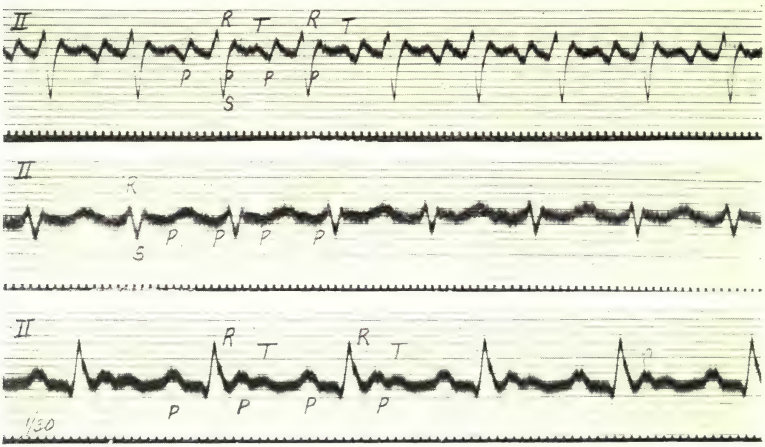


Fig. 65, 66 and 67. Examples of auricular flutter curves. In each 2 : 1 heart-block is present. The auricular rates are approximately 330, 280 and 228, respectively; the ventricular rates, 165, 140 and 114, respectively.

continues runs through *R* and *S*. The next auricular curve commences during the ventricular systole and runs on into ventricular diastole. Both auricular contractions partially coincide with ventricular contractions, and it is to this that the obscurity of this and many other flutter curves is due. Other examples of curves are shown in Fig. 65, 66 and 67. Fig. 65 shows an extremely common type of auricular curve. The auricular contractions are again twice as numerous as the

ventricular. Each auricular beat gives an upstroke at its commencement, but this upstroke is only clearly distinguished where it immediately precedes *R*. It continues to a pointed summit and then the curve slopes away and becomes incorporated in the ventricular complex. The next upstroke coincides with the returning line of *S* ; it comes to a point and slopes away again, but the gradual downstroke is interrupted by a small *T* summit. Thus the auricular tracing as a whole is constituted by a zig-zag line of which the upstrokes are steep and the downstrokes are more gradual.

Fig. 66 exhibits a similar mechanism. The ventricular complexes consist of *R* and *S* deflections. The auricular portion of the curve consists of a uniform wavy line ; one convexity falls between *R* summits, the other falls with them. The general and uniform undulation is clearly visible ; each convexity represents an auricular contraction ; 2 : 1 heart-block is present. The rates of auricle and ventricle are 280 and 140, respectively.

The analysis of Fig. 67 is easier, for here the *P*'s are isolated. Their isolation is due to the lower rates (auricle 228, ventricle 114). The analyses while 2 : 1 heart-block is present are often very difficult and they are sometimes impossible. The true nature of the condition may be revealed only when a higher grade of heart-block becomes established. The first curve of Fig. 72 is not at all dissimilar to that of Fig. 66. The auricular line is a wavy one and there are two convexities to each ventricular systole ; one falls between *R* summits and upon *T*, the other coincides with *R* ; 2 : 1 heart-block is present, the rates being 300 and 150. The same patient demonstrated a higher grade of block upon another day ; the auricular convexities then became clearer, being less confused with the ventricular summits ; the curve is shown in the second strip of Fig. 72 ; 4 : 1 block is present, the rates being 300 and 75. (Similar electrocardiograms are

shown in the first two curves of Fig. 73). The same mechanism is shown in Fig. 68, a curve which has been taken at a faster rate and with a larger excursion. This curve has been marked so as to reconstruct that portion of the auricular line which is obliterated by *R* and *S* ; the regular relation of four auricular convexities to a ventricular systole is made more apparent in this way.

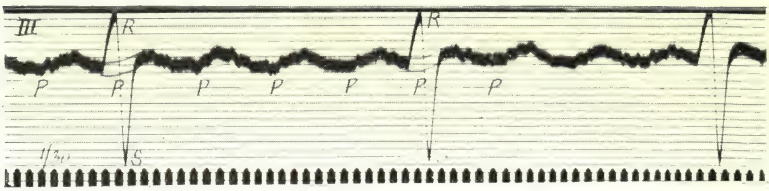


Fig. 68. From a case of flutter, showing 4 : 1 heart-block. Those portions of the auricular complexes which have been obscured by the initial phases of the ventricular complexes have been indicated by reconstruction so as to illustrate the continuity of the auricular oscillations. The standard for this curve is $1\frac{1}{2}$ centimetres to the millivolt.

The continuity of auricular complexes in these curves may be beautifully demonstrated in another way. If the vagal nerves are pressed upon in the neck, the ventricle ceases to beat ; but the auricle continues to contract at its former rate (Fig. 69). The continuously wavy line, each convexity of which stands for an auricular beat, is thus strikingly displayed.

That the active centre in the auricle is ectopic is rendered probable by a comparison of the flutter curves with those of the normal rhythm in the same cases. Examples are shown in the first, second and fourth strips of both Fig. 72 and Fig. 73. In the first strips of Fig. 72 the auricular beat gives a simple convexity. In the last strip, in which the normal rhythm is present, it gives a short summit followed by a depression. In the first two strips of Fig. 73 the auricular beats are of the same shape as those of Fig. 65 ; there is an

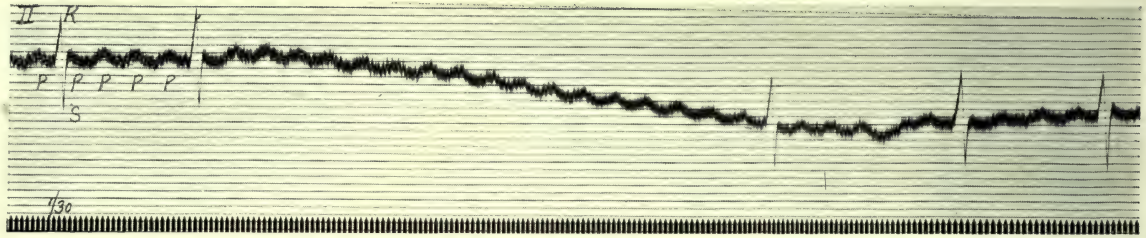


Fig. 69. From the same case, showing the effect of pressure on the right vagus while 4 : 1 heart-block was present. The ventricle ceases to beat, the auricular action shows no abatement.

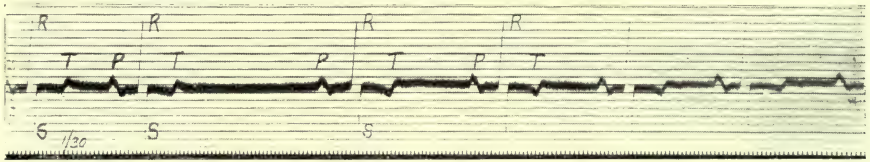


Fig. 70. From the same case after the heart's action had returned to the normal. Pressure on the vagus (right or left) now produced slowing of the whole heart. The left vagus was the one pressed upon in this instance.

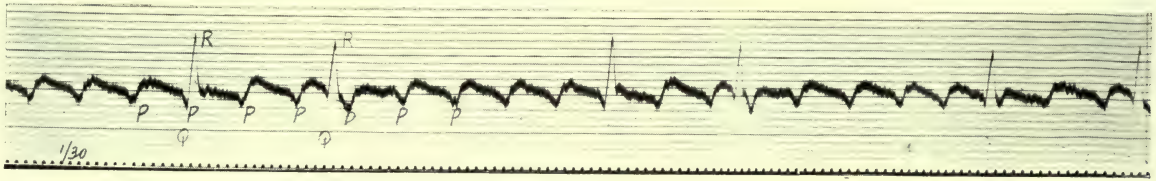


Fig. 71. From a case of flutter on digitalis, showing slow and irregular responses of the ventricle.

abrupt upstroke and more gradual downstroke ; the normal type of auricular complex for this case (lead *III*) is inverted* and split. The ectopic nature of the flutter impulses is also indicated in another manner. Ectopic centres of impulse formation are far less under control than is the normal pacemaker. In the case of simple paroxysms of tachycardia, in which the fast rhythms are known to be ectopic, nerve influences, such as are induced by exercise, emotion, posture or pressure upon the carotid sheath are practically without influence upon the rate at which the impulses are generated. The same statements apply to flutter. Fig. 69 and 70 were taken from the same case and show the effects of pressure on the carotid sheath, during the flutter stage and while the normal mechanism was present. In both cases the vagus has been stimulated, as is shown in the one instance by ventricular stoppage and in the other by slowing of the whole heart with a slight prolongation of the *P-R* interval. Both curves afford evidence of heart-block as a result of pressure, but the essential difference lies in the action upon the auricle. The flutter centre is uninfluenced (Fig. 69) ; the old centre reacts ; the rate at which it builds up impulses is conspicuously decreased (Fig. 70).

Clinical relations of flutter.

Auricular flutter occurs for the most part in elderly subjects and in those in whom there are evidences of cardiac enlargement, myocardial degeneration, and symptoms of an exhaustion of reserve. It appears to result, as do simple paroxysms of tachycardia and auricular fibrillation, from nutritional disturbances in the muscle, which may be a consequence of influenza or rheumatic infections.

* Inversion of *P* in lead *III* does not necessarily signify an ectopic source of impulse formation.

When it develops, it throws a burden upon the heart proportionate to the increase of ventricular rate. When the heart muscle is degenerate and the rate rapid, dilatation, engorgement of the veins, enlargement of the liver and dropsy appear. On the other hand, when it develops in a heart whose muscle has considerable reserve, profound circulatory disturbances and embarrassment are not manifested; but palpitation is common and a retrenchment of reserve power in the response to effort is noticeable. In this manner it disables even those in whom the heart muscle is strong. Fainting attacks are not uncommon in the condition.

The influence which it has upon the life history is not fully known; though it is evident that it is a grave condition when persistent and where the muscle is weak. But a healthy heart may endure it for years, the ventricular rate being maintained at 150 or more per minute. Persistence for months or years seems to be the rule, but flutter also occurs in short paroxysms of a few hours or a few days duration. It may pass spontaneously into fibrillation, but more commonly the change is induced by digitalis administration. The prognosis has at times a good deal in common with that of simple paroxysmal tachycardia and at times is similar to that of auricular fibrillation.* The reaction to treatment should be taken into consideration.

The treatment of auricular flutter is similar to that of paroxysmal tachycardia, except that, having a greater tendency to persist than the latter, the attitude is less expectant and more active. Digitalis and its allies are often very serviceable if given in full doses. In my experience, the ventricular rate can always be reduced, so that 2 : 1 block gives place to heart-block of higher grade. The patient should remain in bed during the treatment. Supposing 2 : 1 heart-block is present originally, then irregularity

* Prognoses which were fully considered in "*Clinical Disorders.*"

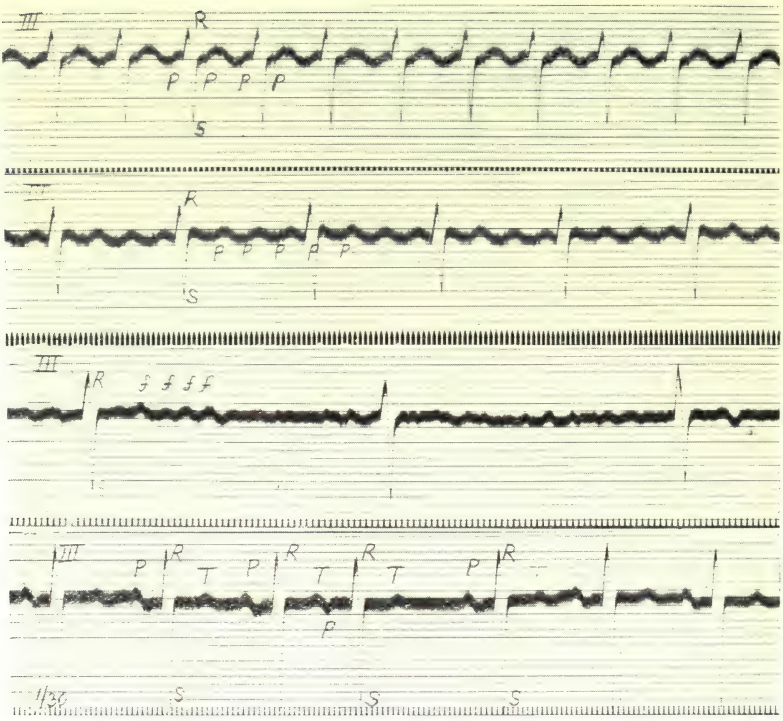


Fig. 72. Four curves from a case of auricular flutter ; showing the effects of treatment. The first curve shows an auricular rate of 300 and a ventricular rate of 150. In the second curve the auricular rate is maintained, but the ventricular rate has been halved (4 : 1 block is present) as a result of digitalis administration. In the third curve auricular fibrillation is seen and it is accompanied by a slow and irregular action of the ventricle. In the last curve the normal rhythm, interrupted by occasional premature contractions of auricular origin, has been resumed.

develops, and this is due to responses at irregular intervals (Fig. 71). Later, and if the drug is continued, the ventricle may become regular again, as a result of the development of 4 : 1 block (Fig. 72 and 73). Under these circumstances the ventricle may be beating at 75 while the auricles persist at 300 per minute, and there may be no evidence, except the electrocardiographic, of the rapid auricular beats. Finally, and

in a large percentage of cases, I find that fibrillation of the auricles appears under the action of the drug (Fig. 72 and 73); and further that, if the digitalis is withdrawn at this stage, the normal heart rhythm is restored (Fig. 72 and 73) and may persist for years. The treatment of auricular flutter by digitalis is often extremely successful and brings with it great improvement of the patient's general condition. With the subsidence of the rapid ventricular rate, cyanosis, engorgement, dropsy and other accompanying symptoms vanish quickly.

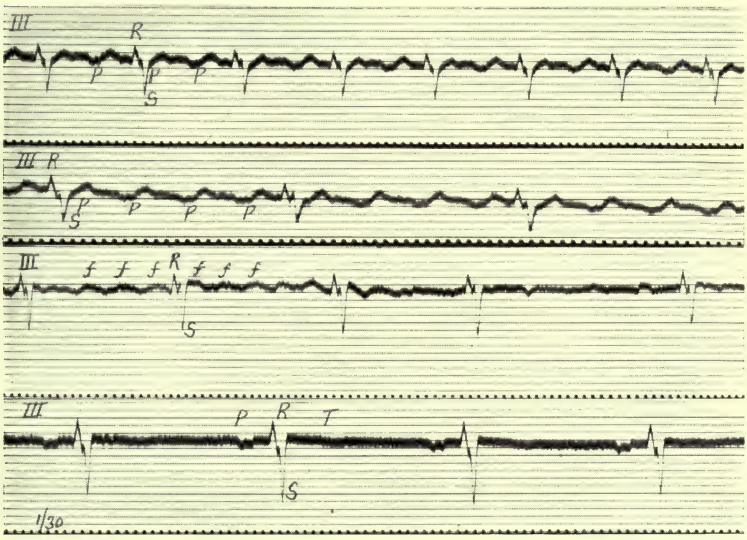


Fig. 73. A similar series of curves to that shown in Fig. 72. They show the effects of digitalis administration upon auricular flutter. In the first curve the auricular rate is 320 and the ventricular rate is 160. In the second the rates are 324 and 81 (4 : 1 heart-block). In the third, the auricles are fibrillating and the ventricle is responding irregularly at an approximate rate of 79 per minute. In the last curve a regular and normal rhythm is established. The inversion of *P* in lead *III* is not significant of an ectopic origin of heart beat in this instance.

Chapter VIII.

AURICULAR FIBRILLATION.

Fibrillation of the auricles, a state in which co-ordinate contraction has ceased in both of these heart chambers and in which the individual strands of the musculature exhibit independent and constant twitching movements, is supremely important amongst all the disorders of heart beat. The muscle of the auricle, though extremely active, has relinquished its function of driving blood into the ventricle; the normal impulses which are transmitted to the ventricle are submerged and replaced by rapid impulses which are derived in a perfectly haphazard fashion from the quivering muscle of the auricle.

The recognition of the disorder is of great consequence, because it is so frequent, because its effects are so profound, and because its reactions to treatment are almost peculiarly its own. It is readily confused with other disturbances when the usual methods of examination are alone undertaken. With very rare exceptions electrocardiography will always detect it. The electric curves of auricular fibrillation are characteristic and afford the only certain means of identifying it. It is recognised by two groups of signs; the first signs result from the changed functional state of the auricles, the second signs depend upon the character of the ventricular responses. We will consider the last first.

When the ventricle responds solely to a fibrillating

auricle, its beats are necessarily of supraventricular origin; they consequently present the normal outlines in electric curves, *R* and *T*, or *Q*, *R*, *S* and *T*, deflections are found. These ventricular complexes have all the same general outline, though the heights of the *R* summits vary in many cases from cycle to cycle. If the ventricular action is slow, then the *R* summits are almost constant in height, but when it is rapid (Fig. 74 and 75) the excursions always vary, and there is then no relation between their heights and the pauses which

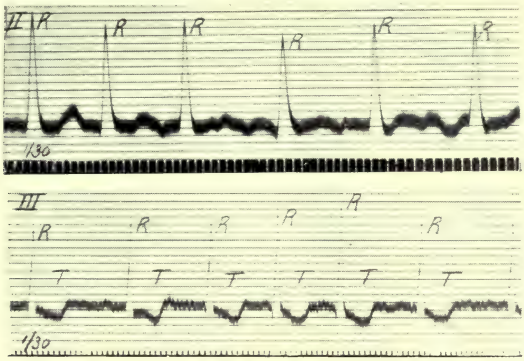


Fig. 74. A curve of auricular fibrillation showing rapid and irregular ventricular response. The height of the peak *R* is variable and is not related to the length of the pause which precedes it. The oscillations are obscured by the rapid ventricular action; that they are present is evident from the changing shape of those parts of the curve which unite adjacent *R* summits. There are no *P* summits.

Fig. 75. A curve of auricular fibrillation, showing rapid and irregular ventricular response, great variation in the height of *R*, and inversion of *T*. Fibrillation oscillations are scarcely seen; the very rapid and minute vibrations are from the body musculature. There are no *P* summits.

precede them. The individual complexes are placed at very irregular intervals. The other group of signs is auricular. The *R* summits have no *P* summits before them, for, as there is no co-ordinate contraction of the auricle, so there is no presystolic auricular representative in the electric curve; but the quivering flesh of the auricle yields pronounced electric waves, oscillations which characterise the majority

of curves. The type of oscillation deserves study; the approximate frequence is 500 per minute; so, if the ventricle beats at 83, there will be about six oscillations to each cycle, or if it beats at 125, there will be about four oscillations to the cycle. Only those oscillations which occupy diastole are prominent; and as a consequence, if the ventricle beats slowly, the oscillations are conspicuous; but if it beats fast, they are often difficult to distinguish.

The oscillations of the first curve in Fig. 80 are very large, and it should be remarked that there is an attempt towards regular disposition, but the individual swings of the fibre *are never quite uniform as in flutter*. When *T* is prominent, as in this curve, it is deformed by oscillations which fall with it. The oscillations are continuous, but in one place (between the third and fourth *R* summits) the amplitude is diminished. This variation is always present, and if the mean amplitude is small, then the oscillations may vanish from place to place in the curves. In Fig. 81 (top curve) they are prominent at the beginning, but towards the end they are barely perceptible. When the ventricle beats fast, as in Fig. 74 and 75, diastole is comparatively short and they are not clearly seen. Their presence is ascertained in such curves by scrutinising the lines which join the *R* summits; these lines have never quite the same configuration from cycle to cycle. The oscillations are in reality quite continuous, but, as in flutter curves (Fig. 65 and 66), fast ventricular action obscures them. They are especially prominent in cases of mitral stenosis (Fig. 80 and 94). In other conditions they are often small; Fig. 76 is an example of such a curve; this electrocardiogram should immediately suggest fibrillation, for the ventricular complexes are all of the supraventricular type, and quite irregularly spaced; furthermore, there are no *P* summits. There are deflections in Fig. 74 which suggest auricular contractions at first sight, but they are inconstant in form and

position and should not mislead, being in reality individual and prominent oscillations which happen to fall in presystole.

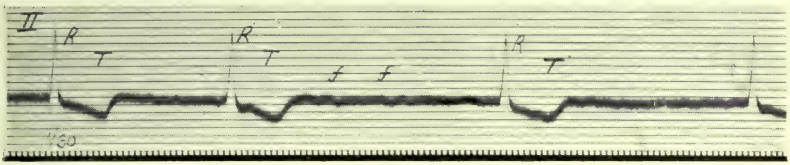


Fig. 76. From a case of fibrillation under treatment with digitalis. It shows irregularity of the ventricle and inversion of *T*. The oscillations are small; *P* does not appear.

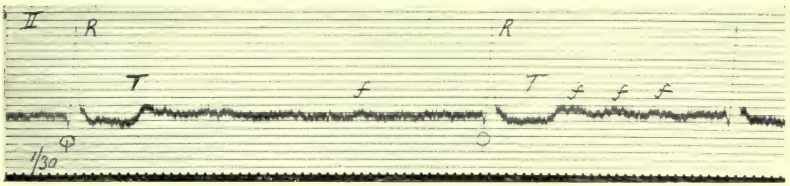


Fig. 77. From a case of fibrillation under treatment with digitalis. The ventricular action is irregular. The diastolic portion of the curve shows two series of oscillations, the one (*f.f.*) due to the fibrillation of the auricle; the other, in which the oscillations are extremely rapid, resulting from tremor.

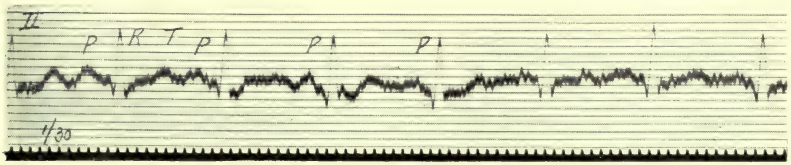


Fig. 78. From a case of exophthalmic goitre, showing a regular heart action. Each ventricular complex is preceded by a summit *P*, but the whole curve is disturbed by oscillations resulting from tremor of the somatic musculature.

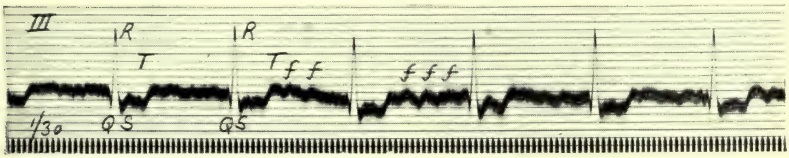


Fig. 79. From a case of auricular fibrillation, under treatment with digitalis. The fibrillation is evidenced by the oscillations *f.f.* and by the disappearance of *P* summits. The ventricular action is regular because complete heart-block is present. The rate is exceptional for a ventricular rhythm, being approximately 90 per minute.

Fig. 77 shows another and distinct form of oscillation, its frequency is very great and it is due to tremor or rigidity of somatic muscles ; it should not be mistaken for that of fibrillation, which has a much lower frequency, though the curve was actually from a case of fibrillation. These minute vibrations are common in fibrillation cases, for the patients are often feeble or actually tremulous, but they may be present when no tremor is detected and may then confuse interpretations. It is often wise to neglect them and to look to the general sweep of the curve. In Fig. 77 they are present throughout and distort the curve ; it is from a fibrillation case, as may be seen immediately from the shape of the ventricular complexes, their arrangement and the absence of *P* summits ; if the curve is regarded with the eyelids almost closed, the fine tremor is no longer distinguished and the slower and inconspicuous oscillations of the fibrillating auricles then become clearer ; these are most prominent after the second ventricular beat and have the characteristic form. Fig. 78 is another and different type of curve, though intended to illustrate the same distinction ; it is from a case of exophthalmic goitre in which there was much tremor. The tremor oscillations are large and coarse, serrating the whole curve and obscuring *P* ; nevertheless *P* or some trace of it may be discovered in each cycle, despite the distortion ; it should be noted also that the ventricular beats are evenly distributed. The cardiac mechanism is normal in this curve. Sometimes, when the somatic muscle tremor is coarse and the patient holds the limbs rigidly or unsteadily, the curves present difficulties and there may be doubt as to whether a given series of oscillations is of somatic or cardiac origin ; under these conditions special leads may profitably be adopted. Small metallic discs are fastened directly to the chest wall with a stiff paste of flour and salt. Convenient points of contact are depicted in the accompanying diagram

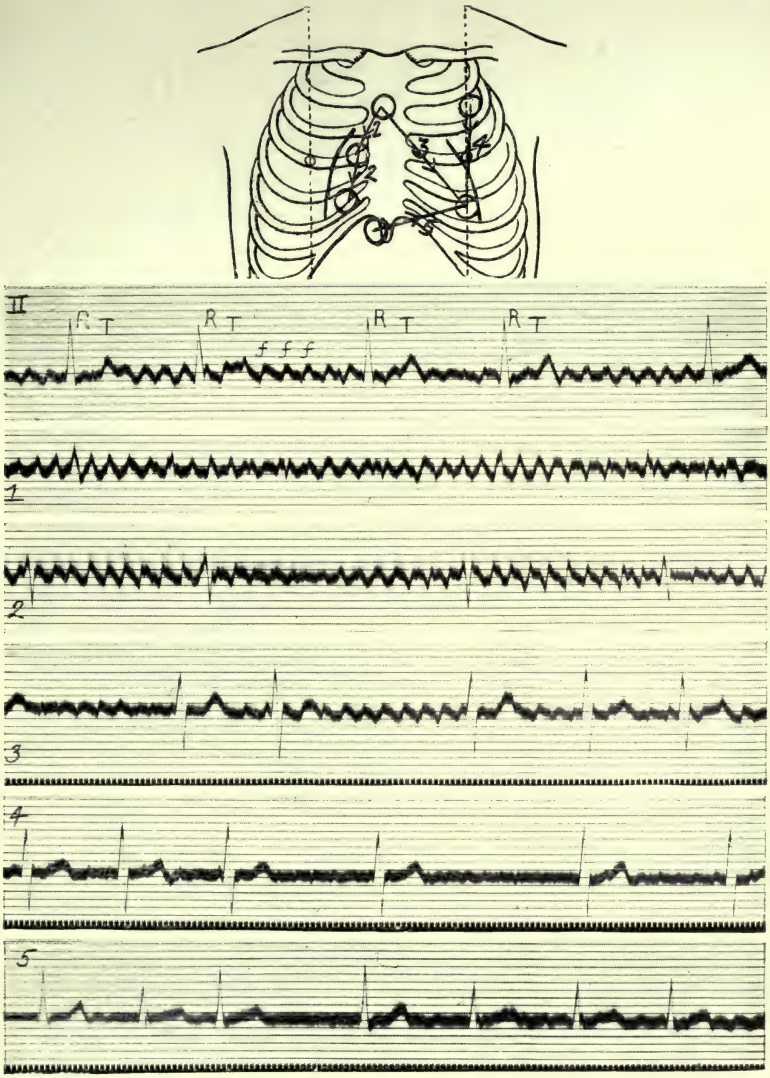


Fig. 80. A diagram of the chest wall showing the special leads (*1* to *5*) used in identifying the oscillations of auricular fibrillation; also six electrocardiograms. The first electrocardiogram is from lead *II*; it consists of irregularly placed ventricular complexes (*R*, *T*) and of large and continuous oscillations (*f.f*). The remaining five curves are from the chest wall. *1* and *2* were taken from the area overlying the right auricle; in these leads the oscillations are maximal and the ventricular complexes are minimal. *3* was taken from an oblique lead covering the whole heart, and it shows both oscillations and ventricular complexes. *4* and *5* were taken from leads along the margins of the ventricles; they show but little sign of the oscillations. From a case of mitral stenosis.

which illustrates the method (Fig. 80). When fibrillation is present and the electrodes lie in the vicinity of the right auricle (leads *1* and *2* of the diagram) the oscillations are maximal, and there is but a trace of the ventricular beats. When they lie in the long axis of the heart (lead *3*) then both the oscillations and the ventricular complexes are conspicuous. Finally, when they lie along the left or right ventricular border (leads *4* and *5*) the ventricular complexes are clear cut while the oscillations are small or absent. The corresponding electrocardiograms are shown below the diagram, the first curve of which is from the customary lead *II* (right arm to left leg). The special contacts analyse this axial lead, breaking it into its auricular and ventricular constituents. The oscillations of fibrillation are readily identified in this manner and their origin in the auricle is clearly indicated. In tremulous subjects, no oscillations are seen in any of the special leads.

The chief features of the electrocardiograms in fibrillation of the auricle are strikingly displayed when a comparison can be instituted between the curves of this condition and those showing the normal rhythm in one and the same case. Fibrillation, though usually a chronic and persistent disorder, occasionally occurs in short paroxysms or, as we have seen, terminates a period of auricular flutter. In such patients the comparison may be made. The curves of Fig. 81 were taken from the same patient within a few days of each other. The ventricular complexes are of similar type in both ; in one they are distributed regularly and are preceded by the usual *P* summits (second curve) ; in the other the spacing is irregular, no *P* summits are discovered, and the whole base line exhibits the oscillations of fibrillation (first curve).

Two groups of signs have been insisted upon, namely the auricular and ventricular, respectively. In the great majority of curves, the signs of both groups are found. We may now

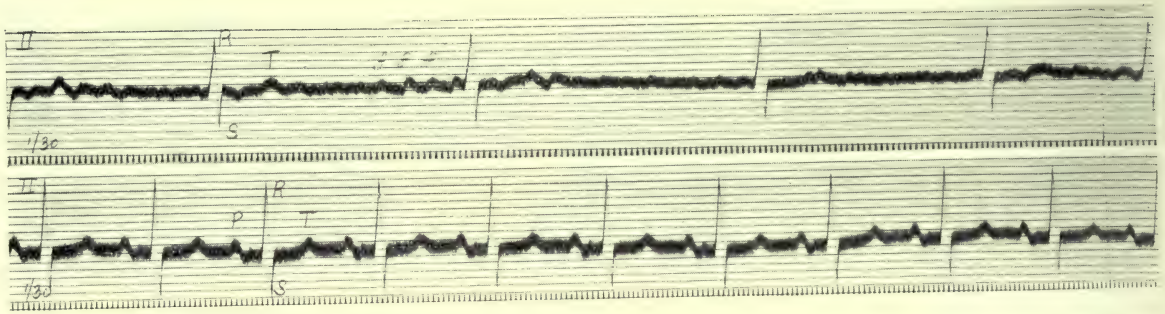


Fig. 81. Two curves from the same case; the upper one shows auricular fibrillation, the lower one displays the normal rhythm. In the first the ventricular action is irregular, there are no *P* summits and oscillations are conspicuous. In the second the action is regular, *P* is prominent and there are no oscillations. Note the similarity of the ventricular complexes in the two curves.

discuss the exceptional curves. It has been said that the oscillations may almost fail; absolute failure, if it occurs, is so rare that it needs little consideration. More important, from the standpoint of diagnosis, is the association of auricular fibrillation with *regular* action of the ventricle. It is also very infrequent, but occurs when the auricular impulses no longer control the ventricle; in other words, when the last named chamber beats independently.

We shall have reason to reconsider this association almost immediately. At the present time, being exceptional, it serves to emphasise the rule, that while auricular fibrillation is present, the ventricular movements are disorderly. Fig. 79 serves to illustrate these remarks; it shows the usual type of oscillation, and no auricular summits *P* are seen, but the ventricular beats have natural outlines and are regular in incidence. It is always necessary that the origin of the oscillations should be confirmed by means of the special leads in these patients.

Fibrillation is the acme of auricular disturbance, consequently it is never complicated by other perversions of auricular rhythm. But the heart which exhibits it may display any other type of disorder. These complications require brief description. And first we may take heart-block. The responses of the ventricle to a fibrillating auricle are naturally rapid and approach 200 per minute. A slower ventricular action is an indication of heart-block, and the degree of heart-block is gauged by the degree of slowing. It is by producing high grades of partial heart-block and by assuaging the original rapidity of the ventricular beats that digitalis and its allies produce their most notable clinical effect. Fig. 76 and 77 were taken from patients fully under the influence of these drugs; the ventricular rates were 47 and 52 when the curves were taken; before treatment they had been rapid. Digitalis heart-block may be complete

(Fig. 79 is an example) and when complete from this cause, the rate of the regular ventricle is relatively high. Heart-block resulting from bundle lesions produces slow ventricular action when the auricles fibrillate, and when complete, the ventricular rates are of the same grade as those found in simple heart-block. Thus fibrillation may be accompanied by ventricular rates which range from 30 to 200, according to the facility with which the auricular impulses pass to the ventricle.

The ventricular complexes in fibrillation are, as has been said, of the supraventricular type, but *T* may show inversion (Fig. 76) and the aberrant forms of beat, described on page 31, may occur, when one or other bundle branch is damaged. The amplitudes of summits *R* and *S* in the three leads have the same significance as they have when fibrillation is absent (see page 24 and Fig. 94).

Premature beats are also seen, they spring from ventricular foci.* An example of this kind is given in Fig. 82; this curve was taken from lead *III* and the diminutive *R* and deep *S* are indications of left ventricular hypertrophy; a single anomalous contraction, a premature beat, is seen towards the end of the figure, and its form in the electric curve is that of a beat arising in the left and apical portions of the ventricle. These premature beats are traced most often in patients who are taking digitalis or the allied drugs; appearing under this circumstance, they suggest relaxation of the treatment. When more frequent, they are urgent messengers that the drug be discontinued; the condition is exemplified by Fig. 83; it is known as "digitalis coupling." Each ventricular complex of the supraventricular type (*R*, *T*) is followed at a close and constant interval by a

* Almost exclusively so; naturally they cannot originate in the auricle, but some appear to originate in the junctional tissues.

Fig. 82. From a case of auricular fibrillation. With a single exception, all the ventricular complexes are of the supraventricular type; the corresponding ventricular contractions are responses to the fibrillating auricle. The premature beat *P.B.* is of different origin, it comes from the apical and left portions of the ventricle.

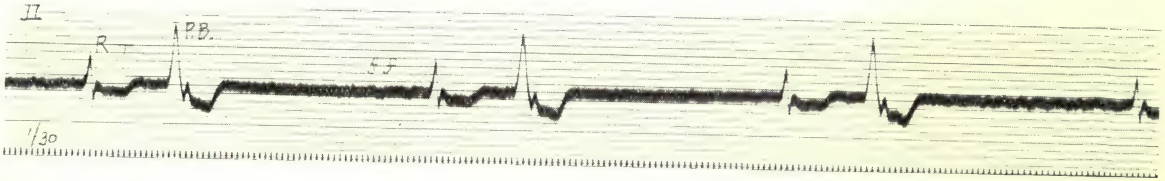


Fig. 83. From a case of auricular fibrillation on large doses of digitalis. It shows the coupling of ventricular beats which speaks of over-dosage. The first complex in each couple is of the supraventricular type; the second is of different form; these last contractions (*P.B.*) are premature and originate in the ventricle. The pauses succeeding the couples are of equal length; complete heart-block was also present in this instance.

complex of anomalous form. In this instance, the form is that of beats coming from the right and basal portions of the ventricle. The ventricular rate is slow; there are no *P* summits, but there are small oscillations during the diastolic periods. The auricle is fibrillating. A continuation of digitalis, when this coupling has declared itself is culpable; it is followed only too frequently by sudden and avoidable disaster.

CHAPTER IX.

SINUS DISTURBANCES AND ALTERNATION.

Respiratory arrhythmia and allied irregularities.

There are a number of closely related heart irregularities, which are due to variations in the rate at which the impulses are generated at the physiological pacemaker. A notable example is an irregularity of respiratory origin (Fig. 84) in which there is a gradual acceleration of rate during the inspiratory phase and a fall of rate during the expiratory phase of respiration. In young subjects it is a normal phenomenon; and during the earlier periods of life, allied irregularities, in which the whole heart participates but in which there is no constant relation to the acts of breathing, are observed (Fig. 85). These irregularities, often grouped under the term " sinus arrhythmia " are of nervous origin and are brought about by alterations of vagal tone. In electrocardiograms they are readily recognised. Each beat of the heart is propagated from the natural pacemaker; the electric curve is consequently formed of physiological complexes, in which the usual auricular and ventricular summits are seen. Irregularity is evident only in the disposition of the beats; the normal sequence of chamber contraction is retained.

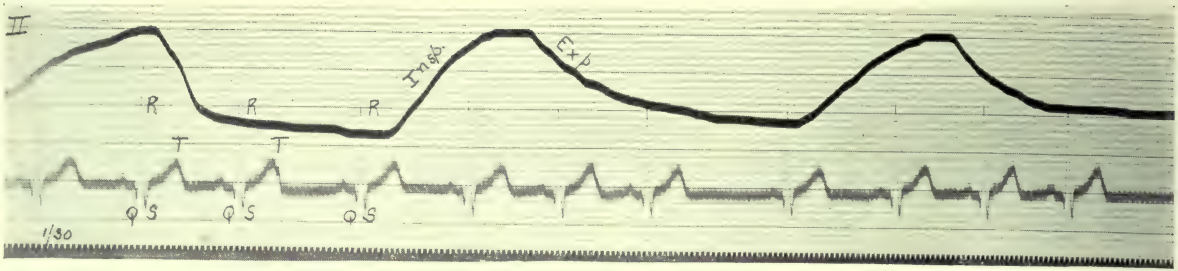


Fig. 84. Electrocardiogram and curve of respiration; showing a sinus arrhythmia. The heart quickens during the period when intrathoracic pressure is low. The whole heart is involved in the irregularity.

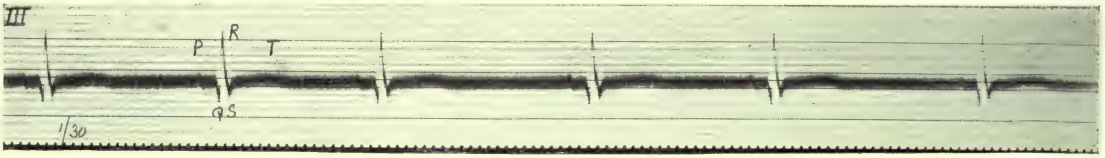


Fig. 85. A sinus irregularity, in which there was no relation to respiration. It was taken from a healthy child, a few hours after birth.

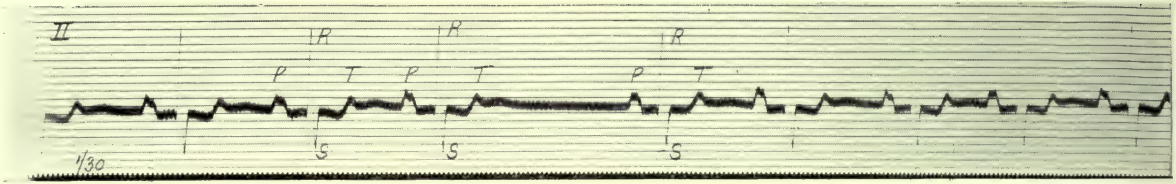
Sino-auricular heart-block.

There is another though infrequent form of irregularity, which is related to those we have just considered. It is termed "sino-auricular heart-block" provisionally. This irregularity is but imperfectly understood and its significance, though apparently inconsiderable, is not fully known. In many patients it is of vagal origin; it is not uncommonly associated with auriculo-ventricular heart-block and like the latter may occur as a result of digitalis administration. It generally manifests itself in one of two ways; by producing intermittences of the whole heart action; or by leading to steep falls of heart rate. When a single heart beat is lost (as in Fig. 86), the length of the longest cycles is usually somewhat shorter than two cycles of the natural rhythm. When the heart rate falls, the passage from one rate to the other is abrupt; there is no transitional period (Fig. 87); and although the slow rate may be almost exactly half the former rate (Fig. 87) yet more commonly actual halving of rate is scarcely attained. Permanent slow action of the whole heart may be due to this mechanism (Fig. 35). It is supposed that these irregularities are due to some hindrance in the passage of the rhythmic impulses which issue from the region of the superior cava, to the main mass of the auricular tissue; but whether this is the true explanation or not remains to be proved.

The electrocardiographic curve of each heart beat presents an auricular and a ventricular complex and these are of the forms known to be associated with a heart beat arising in the physiological centre of impulse formation.

Alternation of the heart.

When alternation of the heart is present, and is displaying itself in the arterial pulse, the electrocardiograms do not necessarily show evidences of it. If the heart is beating



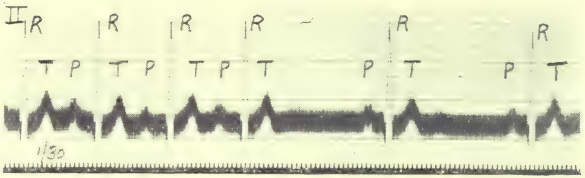


Fig. 86. "Sino-auricular block." An irregularity in which the whole heart is involved. A long pause interrupts what is otherwise an almost regular heart action. The long cycle is nearly, but not quite, twice the length of the short cycle. From a case of mitral stenosis (note the large split *P* summit). The irregularity resulted from digitalis.

Fig 87. "Sino-auricular block" in a case of exophthalmic goitre. There is a sudden and almost exact halving of the rate of the whole heart. Note the prolongation of the *P-R* interval in the last cycle of the fast rhythm.

slowly, and alternation of the pulse is conspicuous, the several ventricular summits and depressions are often of uniform excursion from beat to beat (Fig. 88), nevertheless the alternation may be conspicuous in the electrocardiograms ; it is seen to advantage in Fig. 63. In this figure it is increased after the premature contraction. When alternation accompanies paroxysms of tachycardia, traces of alternation in the amplitudes of *R* and *T* are frequent. The left-hand

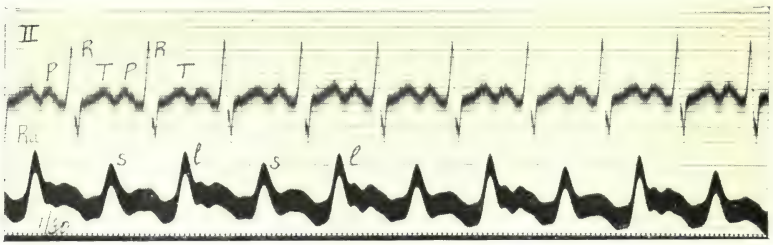


Fig. 88. Simultaneous electrocardiogram and arterial curve. The last shows conspicuous alternation ; but in the electrocardiogram it is not perceptible (see Fig. 62 & 63).

portion of Fig. 62 serves as an illustration ; the alternation is confined to the *R* summits and, though of minor degree, is quite distinct. Curiously enough the alternation in electrocardiogram and pulse is not always parallel ; while the small *R* summit may correspond to the small pulse beat, yet quite as frequently (as in Fig. 62) the large *R* summit corresponds to the small pulse beat. On occasion alternation of amplitude may be present in the electrocardiogram when the pulse fails to show it.

CHAPTER X.

SPECIAL CONDITIONS.

Electrocardiograms in valve lesions.

At the outset, it is necessary to state emphatically that electrocardiography has little to do with valve lesions. The method is essentially one which investigates the state of the muscle ; only, therefore, in so far as valve lesions affect the muscle, do they influence the form of electric curves. Now the degree to which valve lesions upset the distribution of the mass of heart muscle is a question upon which we have no final evidence ; there is always the primary difficulty that whatever may have affected the valves may also have affected the contractile substance. The indiscriminate assignment of hypertrophy of this or that chamber to leakage at this or that valve as a casual agent cannot be too strongly deprecated. However boldly it may be asserted in a given case that preponderance of left-or right-sided hypertrophy is the result of purely mechanical defects, the conclusion in the individual case is often impossible of proof. We know that free aortic leakage may consort with a light or heavy musculature ; we know that, produced under strictly aseptic conditions, reflux at this valve is generally followed by but relatively little muscle change ; nothing is more certain but that some of the largest human hearts are found where no sign of valve defect or heightened pressure has ever been discovered.

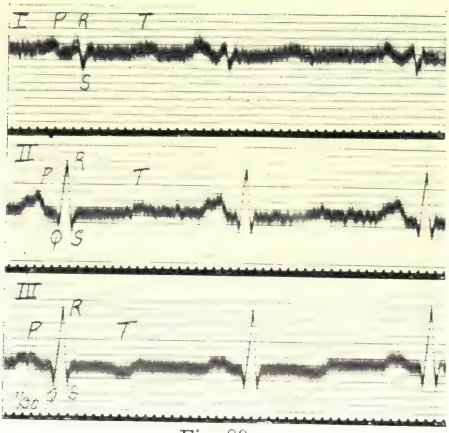


Fig. 89.

Fig. 90.

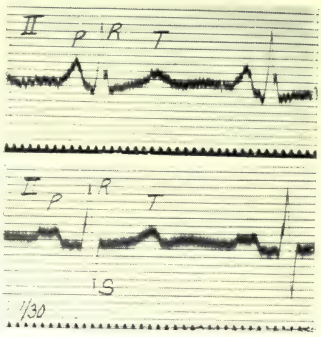


Fig. 91.

Fig. 89. From a case of mitral stenosis. The summit *P* is large, broad and notched in lead *II*. The initial ventricular peaks of lead *I* are small. *R* is tallest in lead *III*.

Fig. 90. From a case of mitral stenosis, showing a tall *P* summit.

Fig. 91. From a case of mitral stenosis, showing a broad and notched summit *P*.

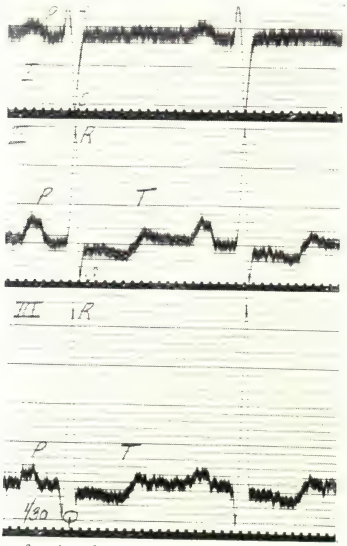


Fig. 92. From a case of mitral stenosis. The summit *P* is large, broad and notched in lead *II*. *R* is of least amplitude in lead *I* and of greatest amplitude in lead *III*. *S* is of greatest amplitude in lead *I* (hypertrophy of right ventricle).

While alteration in the muscle as a sequel to pressure changes cannot be denied, yet it is becoming increasingly evident that this hypothesis has in the past outstripped the facts. No question of cardiac pathology requires more critical revision to-day. Newer and more accurate methods of observation, untrammelled by prejudice, are needed. These remarks are a necessary prelude to those which follow ; if we look for the electrocardiograms which are held to depict hypertrophy of this or that heart chamber in the case of a given valve lesion, the result will often fail to fulfil our expectations.

Mitral stenosis. The electrocardiograms of mitral stenosis are often so characteristic that the valve lesion may be diagnosed from these curves alone. The summit *P* has an exaggerated amplitude, amounting frequently to 2, 3 or even 4 scale divisions (Fig. 89, 90, and 91 and 92 ; and also Fig. 30, 53, 54 and 86) ; it is often broad, flattened and notched in the centre (Fig. 89, 91 and 92). The ventricular complexes generally indicate the presence of relative preponderance of the right muscle. Often this change is shown conspicuously (Fig. 92), *S* being exaggerated in lead *I* and *R* in lead *III*. Sometimes the change is less evident ; small *R* and *S* deflections in lead *I* (Fig. 89) are not uncommon. When the auricles are fibrillating the evidence of right-sided preponderance remains (Fig. 94), and the exaggerated *P* summits are replaced by large oscillations (Fig. 74, 79, 80 and 94). Oscillations of great amplitude (Fig. 80) are never seen except in this condition, so far as my experience goes. When new auricular rhythms, constituting paroxysms of tachycardia, occur in mitral stenosis, the ventricular peaks still suggest right-sided hypertrophy, but the *P* summits no longer occur in their usual forms (Fig. 93). These facts are often helpful in the recognition of the valve lesion when other signs are insufficient. Especially is this the case when, the auricles

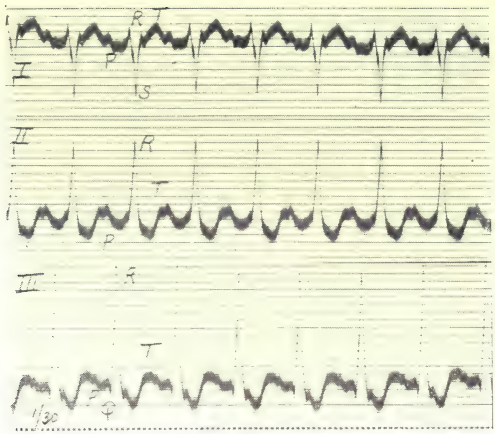


Fig. 93. From a paroxysm of tachycardia in a case of mitral stenosis. Relative preponderance of the right ventricle is indicated; the *P* summits are small because the paroxysm arose in an ectopic auricular focus.

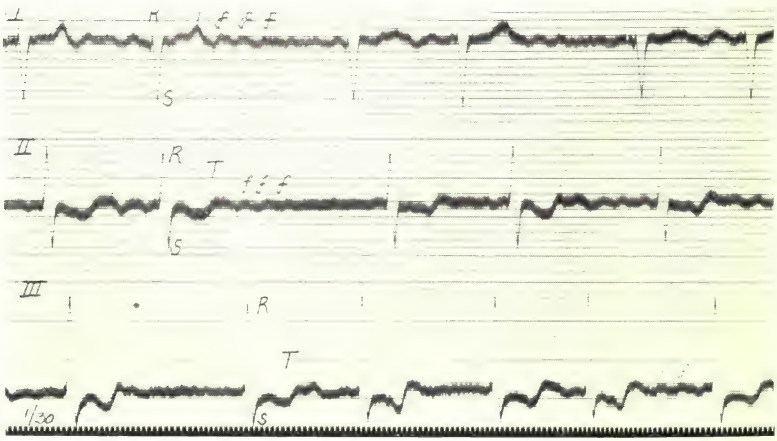


Fig. 94. From a case of mitral stenosis in which the auricles were fibrillating. Relative preponderance of the right ventricle is indicated in the ventricular complexes. The oscillations are prominent, as is usual in mitral stenosis.

being in fibrillation, the presystolic murmur fails or is replaced by a murmur falling in early diastole. The records are helpful in differentiating diastolic murmurs of aortic, pulmonary and mitral origin (Flint's and Steell's murmur).

Aortic disease. The curves of aortic disease are most varied in form; the uncomplicated valve lesion produces little or no alteration. In a dog in which free regurgitation was produced, the electrocardiograms, taken before and some forty days after operation,

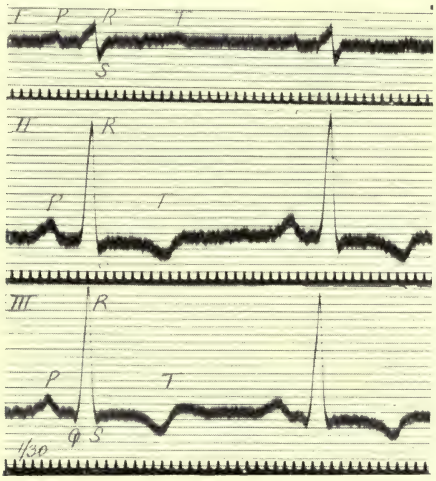


Fig. 95. From a patient in whom aortic regurgitation was present, showing inversion of *T* in lead *II*. The curve is exceptional because it speaks for relative preponderance of the *right* ventricle.

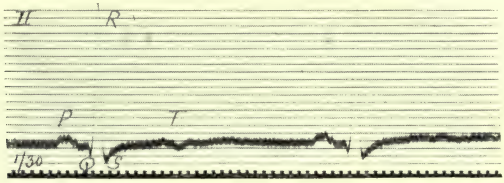


Fig. 96. From a case of aortic regurgitation, showing the flattening of *T* which is common in hearts which have this valve lesion.

showed no appreciable change ; such change as was exhibited was unstable and was no greater than that which often occurred from day to day. It is true that left-sided ventricular preponderance is often indicated in the curves of the human subject (Fig. 19 and 24), but it is not shown with constancy in this condition, and is encountered more frequently when aortic disease is absent. The picture of right-sided preponderance is also compatible with lesions of this valve, though the association is exceptional (Fig. 95). A large excursion of *R* in lead *II* (Fig. 95 and 96), smallness

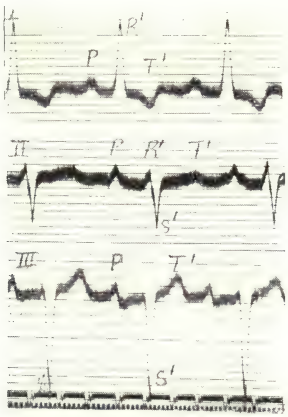


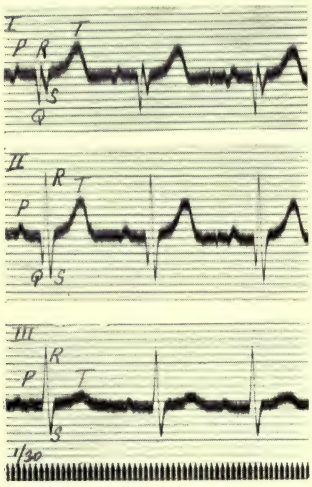
Fig. 97. From a patient in whom aortic regurgitation was present. It serves to emphasise the importance of distinguishing between two series of curves, namely, those which are associated with left ventricular preponderance and those which indicate a deficiency of the right branch of the auriculo-ventricular bundle.

of *T* (Fig. 96) or inversion of the same summit in lead *II* (Fig. 95) are quite common. The absence of constant signs is attributable to the diversity of muscle change and distribution when this valve lesion is present.

The electrocardiograms which indicate functional inefficiency of the right division of the bundle seem to be more than coincidentally frequent in aortic disease ; and occurring

in aortic disease they may be confusing. Fig. 97 serves as an example. Now these curves were taken at a slow rate, and a cursory examination would suggest hypertrophy of the left ventricle. But the initial phases of the ventricular complexes are long, they together exceed a tenth of a second in duration. The inversion of the deflection marked T^1 in lead *I*, the amplitude of T^1 in lead *III* and the notching of the downstroke of S^1 in lead *II* are all compatible with the bundle lesion (compare Fig. 21 and 23). A more unquestionable example of bundle branch defect in aortic disease has been given already in Fig. 23.

Mitral regurgitation. The curves show no constant character.



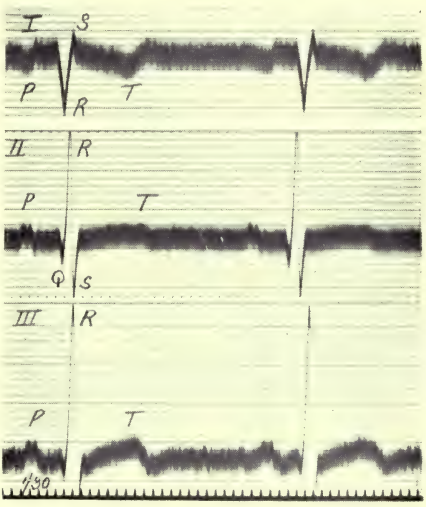
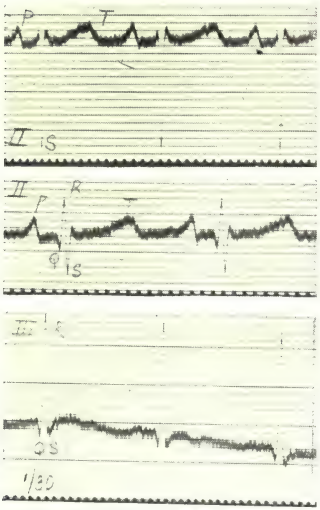


Fig. 98. From a case in which there was conspicuous displacement of the heart towards the right side.
Fig. 99. The curves of a transposed heart. All the summits are inverted in lead *I*.

Congenital heart affections. These curves are often of value. The electrocardiograms, when the heart is transposed, are the most positive signs of the abnormality which we

possess. All the summits of lead *I* are inverted (Fig. 99). A little consideration will make it clear how this change comes about. The lead is a symmetric one, being from the right arm to the left arm. A lead from the *left arm to the right* produces the inverted picture in the normal subject, and the left arm stands to the normal subject as does the right to the subject of transposition. But neither of the other leads is symmetric, and neither of the other leads shows an inverted picture. A lead from the right arm to the



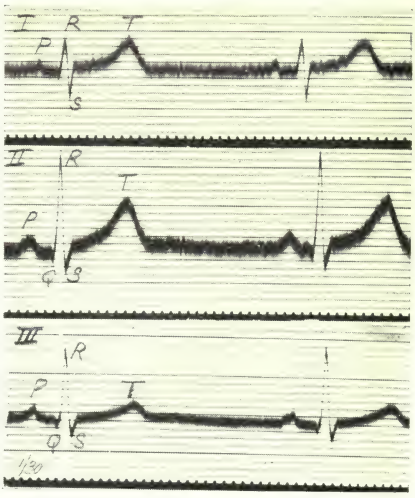


Fig. 100. From a patient in whom there were evident signs of congenital obstruction of the pulmonary artery. Right ventricular hypertrophy is indicated.

Fig. 101. From a patient who presented the signs of a patent ductus arteriosus. The curves are normal.

left leg in any subject gives almost precisely the same picture as does a lead from the right arm to the right leg. So it happens that a lead from the right arm to left leg (lead *II*) in a normal subject is almost equivalent to a lead from the left arm to the left leg in the subject of transposition. Thus,

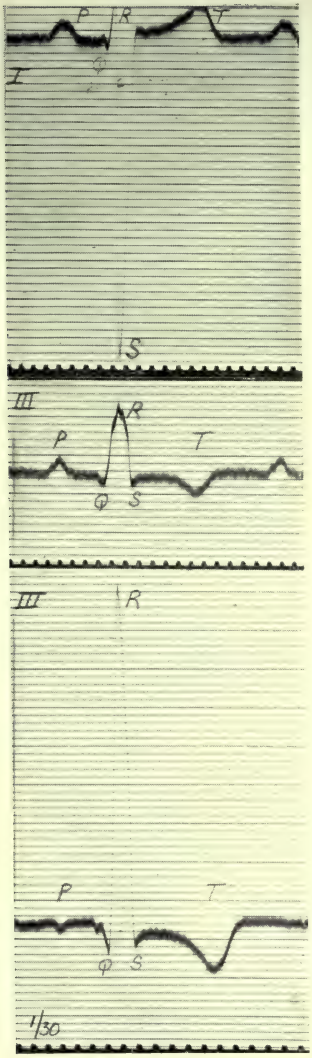


Fig. 102. From a child in whom there were evident signs of congenital pulmonary stenosis. Hypertrophy of the right ventricle is indicated. Note the exaggerated amplitude of excursion in leads *I* and *III*.

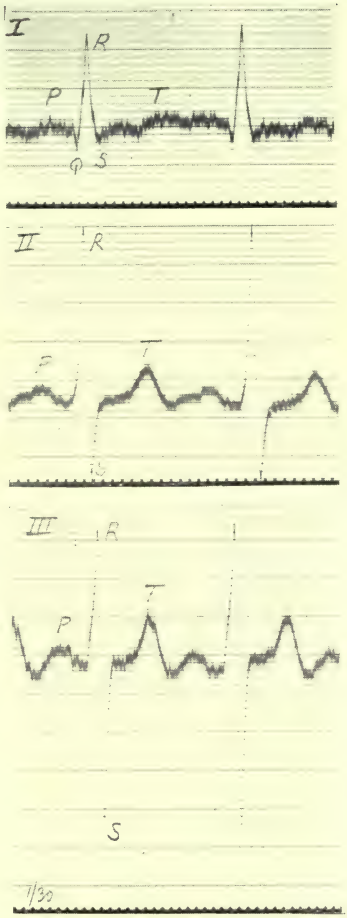


Fig. 103. From a lad with a greatly enlarged heart and congenital malformations. The chief signs were a systolic thrill, palpable over a wide area and maximal at the pulmonary cartilage; a systolic murmur, prolonged into and enforced in diastole, which was maximal in the same region; and a systolic apical murmur. The right carotid artery crossed the trachea. Note the amplitude of excursion in leads *II* and *III*.

in the subject of transposition, while *the curve from lead I is inverted, those of leads II and III replace each other.* If we wish to draw conclusions as to the distribution of muscle from Fig. 100, we must view lead *I* in a mirror, placed above it, and we must place lead *III* between leads *I* and *II*. The value of the transposition curves will be evident; displacement of the heart distorts electrocardiograms, but does not induce these changes. An example of extreme displacement is illustrated by Fig. 98. It was taken from a boy; the heart's impulse was found well to the right of the sternum, and no reason for displacement was apparent. The question of transposition arose, but the electrocardiograms negatived the presence of this anomaly; the chief distortion is in the opening ventricular events of lead *I*.

In those children in whom cardiac enlargement, cyanosis and harsh systolic thrills and murmurs, maximal over the pulmonary cartilage, are the chief diagnostic signs the electrocardiograms generally indicate extreme degrees of right-sided hypertrophy. They are instances of pulmonary stenosis. Fig. 100 is an example of the curves obtained in this condition; Fig. 102 is an exceptional example from a similar case and illustrates another feature of congenital heart curves, namely, extreme amplitude of excursion. Exaggerated amplitude in several leads is in itself a valuable sign of congenital valve or septal defects. Another example, Fig. 103, shows this increase of excursion. Such curves are obtained, so far as I know, only when there is reason to believe that congenital malformation is present. It is impossible to speak at all fully of these electrocardiograms at the present time; we require far more information regarding them, and especially a careful comparison with post-mortem material, but there are indications that in the future they will be our chief means of identifying anomalies of development. If there is one congenital defect in which the electric curves

should be normal, it is in uncomplicated patency of the ductus arteriosus. Fig. 101 was taken from a youth who exhibited a continuous harsh murmur over the pulmonary area. The heart was not enlarged and though engaged in laborious work he had had no symptoms from childhood onwards. The curves present no abnormality.

Renal disease and high blood pressure. While the pictures of left-sided hypertrophy are not uncommon, yet as in aortic disease there are no constant associations.

Exophthalmic goitre. It is said that in this condition *T* is exaggerated. I have examined a number of patients and, apart from the enhanced rate of beat and slight changes which may result therefrom, no definite alterations have been seen (see Fig. 57, 78 and 87).

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SHAW & SONS, FETTER LANE, LONDON, E.C.